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THE PLANT DISEASE REPORTER

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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Crops Protection Research Branch
Plant Industry Station
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THE PLANT DISEASE REPORTER

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LOSSES CAUSED BY APPLE SCAB IN NOVA SCOTIA[1]

I. L. Conners[2]

Abstract

From published estimates of the percentage of scabby fruit found in apple orchards in Nova Scotia and of the volume and value of the crop, it was estimated that in recent years loss from scab amounted to $137,000 a year. To protect their crop, valued at $1,685,000, from the worst ravages of the disease the growers in the same period spent $442,000 a year in spray materials and their application.

INTRODUCTION

Apple scab (Venturia inaequalis) is the most important single disease affecting apples in Canada. In all apple growing districts in this country, except British Columbia, a saleable crop cannot be grown without a fungicide spray program and even in British Columbia the trees in most districts are regularly sprayed for scab.

It is certainly a fact that the commercial production of apples would not be an economic possibility without a well-organized pesticide program for the control of diseases and pests of the apple crop. However, if an assessment of the toll taken by a particular disease or pest is to be made, it is necessary to estimate the cost of the spray program to the grower and the losses still sustained by the industry from failure of such a program to give complete protection. The chief difficulty in making useful estimates is the lack of precise data on the incidence of a disease or pest in a defined area. However, some new information on apple scab in Nova Scotia (2) has permitted an estimate of the losses caused by the disease in that province.

DATA AND ANALYSIS

This estimate of losses caused by apple scab in Nova Scotia is based on data drawn to my attention or supplied by Dr. J. F. Hockey, Plant Pathology Laboratory, Kentville, Nova Scotia, and on figures published by the Dominion Bureau of Statistics.

The critical figures on the percentage of scabby fruit (Table 1, Column i) are contained in a paper by Pickett and Patterson (2) and according to them these estimates are based on an annual survey of 80 orchards that had been sprayed for the control of apple scab.

Dr. Hockey estimates that about half the crop of scabby apples goes into the "cull" grade for juice and the other half into "C" grade for peeling. As figures are available only for the part of the crop that was purchased for processing and the price paid for this portion (Columns v and vi), these figures have been used in estimating the loss occasioned by scab (xiii). The estimated amount (xi) of the scabby crop may be too high, but as the calculated unit value (x) of the clean apples (fresh fruit) seems reasonable it is considered that the gross loss (xiii) occasioned by scab was probably close to the figure in the Table.

The acreage (xiv) in orchard in Nova Scotia has been steadily declining in recent years. In 1949 there were about 1,125,000 trees on 24,000 acres in orchard, in 1951 nearly 900,000 trees on almost 20,000 acres, and in 1954, according to the 1954 census in Nova Scotia, slightly over 700,000 trees, which would mean somewhat under 16,000 acres of orchard. Dr. Hockey also estimated that only about five-eighths of the acreage is regularly sprayed (xv) and that the estimated cost of labor and materials is $36.00 per acre.

The calculations in Table 1 may seem unduly complicated, but if any reliable estimate of the losses caused by scab is to be made it is necessary to determine the approximate value of the clean crop that goes into the fresh fruit trade. The clean crop is comprised of apples not only free from any noticeable scab lesions but also fruits free from evident insect injury, spray injury, and other blemishes and bruises. As already indicated, no figures were available on the "farm value" of the clean crop and it was, therefore, necessary to calculate its

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[2]Senior Mycologist.

Table 1. Estimated losses from apple scab in Nova Scotia, 1948-1955 inclusive. [a]

Year	Scabby Fruit	Total Crop			Processed Crop			Clean Crop			Scab Crop			Orchard Area		Spray Costs
		Volume	Farm Value	Unit Value	Volume	Gross Value	Unit Value	Volume	Gross Value	Unit Value	Volume	Unit Loss	Gross Loss	Total	Sprayed	
	i	ii	iii	iv	v	vi	vii	viii	ix	x	xi	xii	xiii	xiv	xv	xvi
	%	bu.	$	$	bu.[2]	$	$	bu.	$	$	bu.	$	$	Acre	Acre	$
1948	31	2,291	1,926	.84	1,657	617	.37	634	1,309	2.06	710	1.69	1,200	26	16	576
1949	9	3,742	2,316	.62	860	346	.40	2,882	1,970	.68	337	.28	94	24	15	540
1950	29	2,250	1,530	.68	1,485	763	.51	765	767	1.00	653	.49	320	22	14	504
1951	24	1,539	1,185	.77	848	428	.50	691	757	1.10	369	.60	221	20	13	468
1952	16	1,626	1,626	1.00	626	388	.62	1,000	1,238	1.24	260	.62	221	18	11	396
1953	14	1,087	1,489	1.37	680	603	.89	407	886	2.18	152	1.29	196	17	10	360
1954	7	2,157	2,114	.98	934	647	.69	1,223	1,467	1.20	151	.51	77	16	10	360
1955	2	3,250	1,294	.40	1,117	451	.40	2,133	843	.40	65	.00	0	15	9	324
Total		17,942	13,480										2,329			3,528
Average		2,243	1,685										291			442

[a]Except in columns denoting Unit Value, figures are in thousands ('000).

[b]Original data in pounds; to convert into bushels, the original figures were divided by 50.

value by difference.

To make perfectly clear how the calculations were made, the figures for the apple crop for the year 1948 will be followed through step by step. In that year the scabby fruit (i) was estimated to be 31 percent of the crop. The total crop comprised 2,291,000 bushels (ii) valued at $1,926,000 (iii), which by division (iii/ii) gives a unit value of 84 cents (iv). This column is not required in estimating the losses from scab, but it does show at a glance the wide fluctuation in the unit value from year to year. The corresponding figures for the processed crop were: volume, 1,657,000 bushels (v), gross value, $617,000 (vi), and a unit value of 37 cents (vii). As for the clean crop, its volume is the volume of the total crop, 2,291,000 bushels (ii), less that of the processed crop, 1,657,000 bushels (v), or 634,000 bushels (viii), valued at $1,926,000 (iii) less $617,000 (vi), or $1,309,000 (ix) with a unit value (ix/viii) of $2.06 (x). The volume of the scabby crop is 31 percent (i) of the total crop of 2,291,000 bushels (ii), or 710,000 bushels (xi). The unit loss of value on account of scab is the unit value of the clean crop, $2.06 (x), less that of the scabby crop, 37 cents (viii), or a loss of $1.69 per bushel (xii). As the scabby crop in 1948 is estimated at 710,000 bushels (xi), the loss caused by scab is placed at $1,200,000 (xiii). In the same way the loss caused by scab was calculated for the other years. It may be noted that in 1955, so little fruit remained on the trees after hurricane Edna, that the floor price paid by the producers constituted the farm price for the whole crop. Under these circumstances, freedom from scab was unimportant.

RESULTS AND DISCUSSION

For the 8-year period 1948-1955, loss from scab is estimated to have been $2,329,000 or about $291,000 per annum. Over the same period the estimated outlay for spray materials and their application was $3,528,000, or $442,000 per annum. Accordingly apple scab in Nova Scotia directly or indirectly cost the growers $733,000 per annum. No account has been taken of the ravages of insects and the cost of controlling them. These results confirm the earlier estimates of Hockey (1). For 1925, one of the worst "scab" years in the history of the apple industry in Nova Scotia, he estimated the loss to the growers of the province to have been at least $750,000. According to him, the 1926 crop was less than half the normal yield, a reduction in part attributable to the lessened vigor of the trees as a result of the heavy scab infection the previous year. Without considering this factor, he still estimated loss from scab in 1926 at $400,000.

It should be pointed out that the decline of the orchard acreage in Nova Scotia is due chiefly to other causes than the losses from apple scab and the cost of the control program. Before 1939, there was a fairly ready sale in the United Kingdom for the culinary varieties commonly grown in Nova Scotia and packed for export in barrels. When after the war limited export was again possible to Great Britain, the demand was confined to boxed dessert varieties, a speciality of the British Columbia growers.

Many of the orchards were also past their prime. Various assistance schemes financed wholly or in part by the Canadian government were organized to remove the overage trees. In 1951, some 289,000 trees were destroyed, bringing the total removed since the war to over 800,000. In recent years also there has been considerable replanting of more suitable varieties so that although the total acreage has been reduced, the opinion is that "the foundation has been laid for a more stable and profitable apple industry" (3) in Nova Scotia.

Nevertheless, when the cost of protecting their orchards and the losses from scab despite this program are considered in relation to the returns realized by the growers it is evident that scab is a disease of paramount importance in apple production. These facts emphasize the value of the work at last underway to seek out sources of resistance and to develop scab-resistant varieties. In the meantime, the plant pathologists must continue the important task of discovering more efficient fungicides and searching for ways and means of improving their application and timing. It is to the advantage of the individual growers to follow these developments closely and adopt improvements promptly in their own orchards.

It would be a matter of great interest if other workers would secure the basic information necessary in their districts so that eventually estimates of scab losses in other important areas of apple production might be ascertained.

I wish to acknowledge the encouragement and advice of Dr. Hockey and of Mr. J. M. Gray of the Dominion Bureau of Statistics, in the preparation of this paper.

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1. HOCKEY, J. FRED. 1927. Apple scab. Canada Dept. Agr. Pamphlet 82 n. s.
2. PICKETT, A. D., and N. A. PATTERSON. 1955. Some observations on the 1955 spray programme. In Nova Scotia Fruit Growers' Assoc. Ann. Rept. 92: 113-119.
3. Tree Removal Committee Report, 1952. 1952. In Nova Scotia Fruit Growers' Assoc. Ann. Rept. 89: 54.

BOTANY AND PLANT PATHOLOGY DIVISION, SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE, OTTAWA, ONTARIO

REDUCTION IN YIELD OF COTTON CAUSED BY DISEASES IN 1957

Compiled by the Cotton Disease Council, Committe on Disease Losses: Harlan E. Smith, Chairman; A. L. Smith, W. E. Cooper, Leonard Lett.

The accompanying tabulated summary of the 1957 Cotton Disease Loss Estimates is the sixth report submitted by the committee on cotton disease losses. As in previous years, the report emphasizes the need for continued and increased funds for phytopathological research and extension.

The summary was compiled as before, from 44 estimates received from cooperators in the States reporting. The accuracy of the estimates is highly reliable, since most of the same cooperators have contributed for the last 6 years and, as a consequence, their methods and techniques are becoming uniform. The committee feels that considerable credence can be placed in the report.

The committee wishes to acknowledge the help of all who contributed to the report and to solicit their further cooperation in making the cotton disease loss summary a continuing success.

The list of cooperators follows:

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Table 1. ESTIMATED PERCENTAGES OF REDUCTION IN 1957 COTTON YIELD CAUSED BY DISEASES.

Disease	Calif.	Ariz.	N. Mex.	Tex.	Okla.	Mo.	Ark.	La.	Miss.	Ala.	Ga.	S. C.	N. C.	Tenn.	Bales Lost	Percent Loss
1. FUSARIUM WILT *Fusarium vasinfectum*	--	--	--	0.19	0.2	8.0	2.5	3.0	3.0	1.0	2.5	1.0	1.0	0.5	148,711	1.11
2. VERTICILLIUM WILT *Verticillium albo-atrum*	3.6	3.44	5.5	0.8	2.5	2.5	4.0	tr	1.0	tr	--	--	0.4	2.0	239,742	1.78
3. BACTERIAL BLIGHT *Xanthomonas malvacearum*	tr	tr	0.3	2.84	0.3	7.0	2.0	0.3	3.5	0.5	1.5	2.0	0.5	0.75	233,067	1.74
4. ROOT ROT *Phymatotrichum omnivorum*	tr	3.16	0.1	2.55	0.3	--	tr	tr	--	--	--	--	--	--	140,845	1.05
5. ANTHRACNOSE BOLL ROT *Glomerella gossypii*	--	--	--	0.1	tr	10.0	--	tr	0.5	0.5	0.45	—	0.3	5.0	74,461	0.55
6. SEEDLING DISEASES *Rhizopus*, etc.	2.75	2.42	2.5	2.03	1.5	1.1	2.0	2.5	2.0	1.0	5.0	10.0	3.0	1.0	322,738	2.41
7. ASCOCHYTA BLIGHT *Ascochyta gossypii*	--	--	--	0.15	--	1.0	tr	--	--	--	0.15	--	0.2	0.3	12,382	0.09
8. BOLL ROTS *Rhizopus*, etc.	1.10	2.30	tr	0.52	2.0	1.0	8.0	3.5	2.5	4.0	1.0	1.0	1.5	0.5	275,961	2.06
9. ROOT KNOT *Meloidogyne* sp.	2.0	2.3	2.0	0.22	0.3	2.0	tr	tr	0.5	3.5	2.5	5.0	2.0	0.75	146,674	1.09
10. OTHERS	tr	0.1[b]	0.4	0.24	0.5[a]	--	--	2.0[c]	--	--	0.5	--	0.3[d]	0.1	16,432	0.12
TOTAL PERCENT LOSS	9.45	13.72	10.80	9.64	7.6	32.6	18.5	11.3	13.0	10.5	13.6	18.0	9.2	10.9		12.0
TOTAL BALES LOST	156,543	127,213	30,269	414,741	23,852	103,991	263,312	50,321	146,781	62,765	61,388	75,731	24,319	55,050	1,586,276	
YIELD IN BALES 1957[e]	1,500,000	800,000	250,000	3,900,000	290,000	215,000	1,160,000	395,000	1,290,000	535,000	390,000	345,000	240,000	450,000	11,760,000	

[a]Various fungal leaf blights.
[b]Crazy top and rust (*Puccinia stakmanii*).
[c]Fusarium wilt.
[d]Miscellaneous foliage diseases.
[e]Nov. 1, 1957 BAE Estimate.

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COTTON DISEASE COUNCIL, COMMITTEE ON DISEASE LOSSES

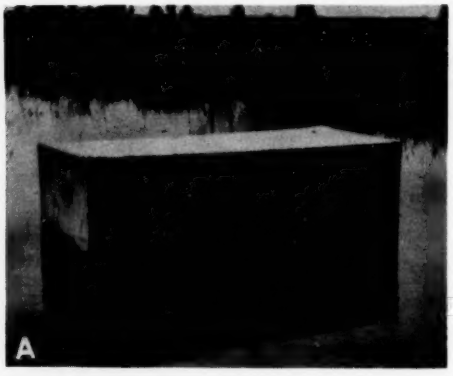
A SIMPLE FIELD INOCULATION TECHNIQUE

J. D. Panzer[1] and R. D. Beier[2]

The use of a tent for inoculation of test plants in the field is a technique that has been used in plant pathology for many years. However, many of the various techniques employing an inoculation tent are inadequate because of time, loss of inoculum, et cetera. An easy field inoculation technique using an inoculation tent and dry inoculum is described below.

A rectangular frame was constructed of 1/2 inch tubular steel in such a way as to measure 6 feet in length, 3 feet in width, and 2 1/2 feet in height. The frame was covered with unbleached muslin on the top and sides, with the bottom left uncovered. An area corresponding to 1/2400 acre was encompassed by the bottom of the tent. The height of the tent may be altered according to the size of the test plants. A hole was then made near the top of each end of the tent to insert the nozzle of a duster. The finished tent is pictured in Figure 1 A.

A dry inoculum-talc or inoculum-perlite mixture may be introduced into the tent by use of a duster such as a small J & P midget duster[3] which can be purchased in many local garden stores. The duster is shown in Figure 1 B. For operation, only the tip of the duster need be inserted into the hole in either end of the tent, while a small cork or cloth plug is used to stopper the hole in the opposite end. This process is repeated on the other end of the tent. The method of dust application is pictured in Figure 1 C.



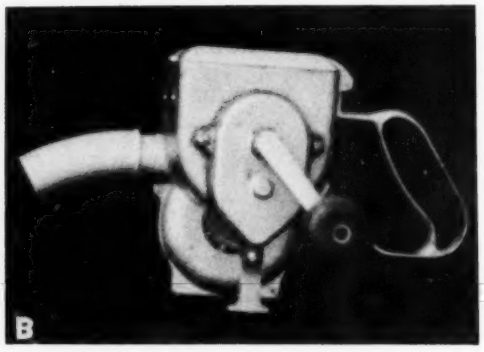


FIGURE 1. Tent inoculation apparatus. A -- Tent, B -- Duster, C -- Application of inoculum. United States Army photographs.

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[3] Manufactured by Jackson and Perkins, Newark, New York.

A definite amount of inoculum may be dusted into the tent by regulating the flow of material from the duster with respect to a definite number of turns of the handle. In the authors' work, 10 turns on either end of the tent were used. Calibration may be made by placing the tent over a large piece of paper marked with one-foot squares. The diluent to be used is then placed in the duster and dusted into the tent according to a known number of turns of the duster. The average deposition per one square foot is calculated after weighing the amount of diluent deposited on each square foot on the paper. When the amount of diluent deposited per number of turns of the duster is known, the percentage of inoculum needed in the inoculum-diluent mixture for a definite amount of inoculum per square foot or per acre is easily obtained.

Small stakes can be placed at each corner of the tent to mark the inoculation site after the tent is removed.

This method has proved very successful for a number of years and although a dry perlite-inoculum mixture was used, this method, with slight modification, should lend itself to various diluents, liquid or dry. Other methods, such as placing a known amount of inoculum into a duster and emptying the duster into the tent, may be used; however, by the present technique, various predetermined amounts of inoculum can be applied without reloading, merely by turning the handle of the duster a definite number of times.

Typical results for infection of rice with three rates of Piricularia oryzae are presented in Table 1.

Table 1. Average number of lesions per foot of row of two rice varieties inoculated with Piricularia oryzae at three rates.

	Grams per acre of spore material		
Rice variety	20	2	0.2
CI 8970	16.7[a]	10.84	6.42
Zenith	2.64	0.15	0.12

[a] Average number of lesions of six tests, three readings per test.

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THE EFFECT OF SEED TREATMENT OF COTTON WITH THIMET, A SYSTEMIC INSECTICIDE, ON RHIZOCTONIA AND PYTHIUM SEEDLING DISEASES

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Summary

Treatment of cotton seed with Thimet 44D, a systemic insecticide, following treatment of seed with one of the mercurial fungicides, Ceresan 200, Ceresan M-2X, or Panogen, under certain environmental conditions has resulted in poor plant stands in the San Joaquin Valley of California. In greenhouse tests neither delay in germination nor decrease in percentage of germination was apparent when cotton seeds treated with Thimet or with Thimet in addition to Ceresan 200 were planted in steamed soil. In soil infested with Rhizoctonia solani Thimet as a seed treatment increased the percentage of emergence and appeared to be fungitoxic, a property not previously recognized for this compound. In soil infested with Pythium debaryanum, Thimet did not increase the percentage of emergence. In non-sterilized soil containing both a Pythium sp. and Rhizoctonia solani, the percentage of emergence of cotton seed treated with both Ceresan 200 and Thimet was much less than from seed treated with Ceresan 200 alone. Treatment of seed with a Thimet-captan mixture or with Thimet following an initial seed treatment with captan has induced a satisfactory stand of cotton in non-sterilized soil.

Thimet (O, O-diethyl S-(ethylthiomethyl) phosphorodithioate) is a systemic insecticide which is being investigated for possible use as a seed treatment to control phytophagous insects or spider mites on seedling plants. This compound has received attention experimentally on a number of crops and has been used commercially to a limited extent as a cotton and alfalfa seed treatment. For purposes of treating seed, Thimet is formulated as a 44% material on activated charcoal (Thimet 44D).

During the spring of 1957, many stands of cotton, obtained from seed treated with Thimet, were much poorer than those grown from seed not treated with this compound. In all cases seed had been previously treated with a fungicide, usually Ceresan 200 (ethyl mercury 2,3 dihydroxy propyl mercaptide, 6% and ethyl mercury acetate, 1.3%, Ceresan M-2X (N-(ethylmercuri)-p-toluene sulfonanalide), or Panogen (cyano(methylmercuri)guanidine). As a consequence of these stand failures, the effect of Thimet on cotton seed germination in steamed, pathogen-free soil and in soil naturally or artificially infested with seedling disease fungi has been investigated.

Since some of the information gained in these studies may have practical application in the immediate future, a résumé of the work is reported here and will be followed by a more detailed report in another journal.

Acid delinted Acala 4-42 cotton seed (95 to 98 percent germination) was treated with Thimet 44D at a rate of 8 pounds per 100 pounds of seed. Seed was first sprayed with a sticker (2% methyl cellulose in water) at a rate of 3 quarts per 100 pounds of seed. The Thimet 44D was applied immediately followed by agitation of seed and insecticide in a glass jar for 3 minutes. Seeds were treated with the liquid mercurial fungicide, Ceresan 200 (1 ounce per 100 pounds of seed), by pouring the proper quantity on the inside surface of a glass jar (about twice the volume needed for the seed) followed by agitation of seed and fungicide for 3 minutes. Captan 75W (a wettable powder) was made into a slurry and sprayed on the seed which was then agitated for 3 minutes.

In most of the tests an attempt was made to simulate cool planting season conditions by incubating flats of soil after planting in a 64° F cold room until the plants began to emerge. The flats were then removed to a greenhouse where the air temperatures varied from 70° to

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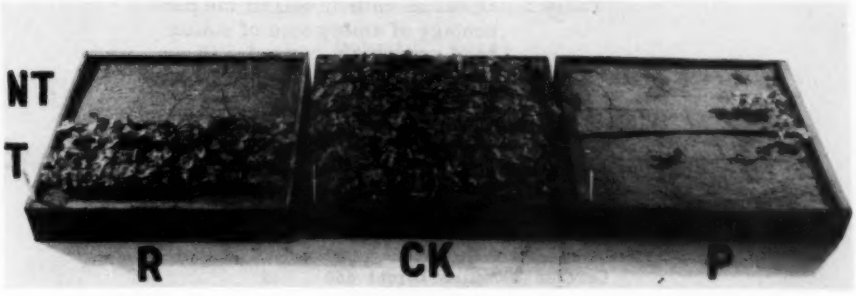

FIGURE 1. The effect of seed treatment of cotton with Thimet 44D on emergence of cotton in Rhizoctonia- and Pythium-infested and non-infested soils. NT -- no seed treatment. T -- seed treated with Thimet 44D at rate of 8 pounds per 100 pounds seed. Flat R, infested with Rhizoctonia; flat CK, non-infested; flat P, infested with Pythium debaryanum.

90° F.

In the tests conducted in steamed soil, neither the percentage nor the velocity of emergence was visibly affected by Thimet as a seed treatment alone or when added to seed previously treated with Ceresan 200. A slight marginal burn of the cotyledonary leaves was the only symptom observed on plants from Thimet-treated seed.

Seed treated with Thimet was protected against Rhizoctonia solani when planted in artificially infested, previously steamed soil (Figure 1). For instance, in one test 93 percent of the plants emerged from Thimet-treated seeds but only 31 percent emerged from the nontreated. After 2 weeks, 74 percent of the plants from treated seeds were healthy, but from nontreated seeds, none were healthy. In another test, Thimet-treated seeds were planted in steamed soil. Two weeks later the plants were inoculated with Rhizoctonia solani. These plants were protected from the Rhizoctonia seedling disease for 7 to 10 days longer than inoculated plants grown from nontreated seed. Eventually, however, all plants were killed by Rhizoctonia.

To test the possible systemic fungitoxic properties of Thimet, sections of roots, cotyledons, stems, and leaves, collected from plants 2 weeks old grown from Thimet-treated seeds were placed on potato dextrose agar on which Rhizoctonia solani was growing. No inhibition of growth of Rhizoctonia occurred. This indicated that the fungitoxic component of Thimet against Rhizoctonia was either not systemic, not present in sufficient quantities in these plant parts to inhibit the growth of this organism, or not diffusible in agar.

When Thimet-treated and nontreated seed were compared in soil artificially infested only with Pythium debaryanum, a nearly total pre-emergence seedling loss occurred (Fig. 1). Thimet did not appear to possess any fungitoxic properties as a seed treatment against this fungus.

The tests to be reported in the remainder of this paper were conducted in non-sterilized soil (3 parts sandy clay loam derived from granite, 1 part peat moss, and 1 part sandy silt). Isolations from diseased seedlings indicated a high inoculum potential of a pathogenic Pythium sp. and a low inoculum potential of Rhizoctonia solani.

Several experiments consistently showed a much lower percentage of emergence from seed which had been treated with both Ceresan 200 and Thimet than from seed treated with Ceresan 200 alone (Table 1).

When captan was mixed with Thimet at either 4 ounces or 1.6 ounces active ingredient per 8 pounds of Thimet and used as seed treatments, a much higher percentage of emergence occurred as compared with that from Thimet or Thimet-Ceresan 200 treated seed (Table 2). When these trials were repeated, the same trends in percentages of emergence resulted. The percentages of healthy plants have been more variable and not always as high as in the test just described.

Table 1. Effect of Thimet 44D on the percentage of emergence of cotton seed previously treated with Ceresan 200 when planted in non-sterilized soil.

Treatments	Total emergence
	%
Ceresan 200	61
Ceresan 200 plus Thimet 44D	5
Thimet 44D	5
No treatment	5

Table 2. Effect of mixing captan with Thimet 44D on percentage of emergence of cotton seed planted in non-sterilized soil.

Treatments	Fungicide per 100 lbs. seed	Total emergence	Healthy survivors (14 days after planting)
	oz.	%	%
Captan plus Thimet	4.0	96	82
Captan plus Thimet	1.6	98	73
Captan	4.0	61	33
Thimet	-	10	0
Ceresan 200 plus Thimet	1.0	3	0

Captan as a slurry seed treatment (1- or 2-ounce dosages of 75% Ortho-Seed Guard per 100 pounds) prior to application of Thimet has been in most cases equally as successful as the captan-Thimet mixture in increasing the percentage of emergence.

Neither chloranil (tetrachloro-p-benzoquinone) (commercially Spergon) nor copper carbonate were effective seed treatments when mixed with Thimet 44D at dosages of 4 and 8 ounces of active fungicide per 8 pounds Thimet 44D.

It appears that either Thimet or the highly adsorptive carrier inactivates the Ceresan 200 seed treatment. Since Thimet is a systemic chemical, physiologic change in the susceptibility of the seedling to Pythium should be considered. This does not appear to be as likely as the previous hypotheses, since the Thimet-captan treatment was more effective than the Thimet-Ceresan 200 treatment. Studies concerning these possibilities are in progress. Other seed treatment fungicides in combination with Thimet and the effects of Thimet carriers other than activated charcoal are being investigated.

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STUDIES ON THE CONTROL OF VERTICILLIUM WILT OF PEPPERMINT WITH CBP-55

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Abstract

Satisfactory commercial control of Verticillium wilt of peppermint was obtained as evidenced by the total plot areas free from infection. In the 0-treated plots 61.4 percent of the area was estimated to be free from infection while 75.1, 79.1 and 85.4 percent of the 40, 80, and 120 gallon per acre plots, respectively, were free from infection.

INTRODUCTION

Control of Verticillium albo-atrum Reinke & Berth. presents a difficult problem which is aggravated by the existence of the pathogen within the host and its survival in the soil. Control measures, apart from utilization of genetic resistance, must be directed at the soil, and to this end considerable effort is being expended.

Green and Luck (1) conducted preliminary screening of the vapor phase of more than 30 potential soil fumigants against mycelial, spore, and sclerotial stages of V. albo-atrum. The two most effective materials were found to be CBP-55 and CPD-162. Tests in muck soil under greenhouse conditions showed that concentrations 2X and 3X above that recommended for mineral type soils were necessary to give comparable results. Field plots incorporated three levels of fumigant application and three sources of planting stock. The results, based on percentage of infection and yields in green top weight, demonstrated that CBP-55, at rates equivalent to 80 gallons per acre, gave highest yields and lowest infection percentages.

METHODS

Field tests were conducted by the author on the William Gehring Farms at Newland, Indiana (Jasper County). The area was divided by a random method into eight, 20 ft. x 50 ft. individual plots. Four dosage levels were employed, 0, 40, 80, and 120 gallons per acre. Each dosage was replicated in two plots.

In this investigation the split application method was employed. Application was made at 2-week intervals and at a depth of 6 inches. A Maclean fumigun (Model #470-2A) was utilized to inject the fumigant. Injection points were 1 foot apart and the fumigation rows were 1 foot apart.

The disease indices were secured by the following procedure. A 40-inch wooden quadrant was divided into four equal quadrates. By this arrangement, the two quadrates on either side of the quadrant were separated by the center of the row of peppermint. The rows of mint were 40 inches apart and the quadrant overlapped 20 inches on either side of the row.

Each 50-foot row was divided into 15 quadrants (a total of 60 quadrants per plot). The quadrants were assigned numbers in the range of 1 to 60 and by a random method 10 quadrants were chosen for each 2-week interval disease index. The quadrants were chosen so that two quadrants were at both ends of the four rows and six quadrants were within the area of 40 inches from the row ends.

RESULTS AND DISCUSSION

For the purposes of this investigation the following symptoms were used as indications of the disease in mint: a) retarded elongation, b) shoot and leaf asymmetry, c) blanching of the leaves, and d) wilting and defoliation. Symptoms a, b, and c appeared prior to July 15, but after July 2. Wilting and defoliation occurred around the first of August.

Knowing the total number of quadrants per plot, it was possible to estimate the percentage of total infection or the total area free from infection for each plot on any given date. The results (Figure 1) indicate that it might be possible to obtain a satisfactory control of the wilt

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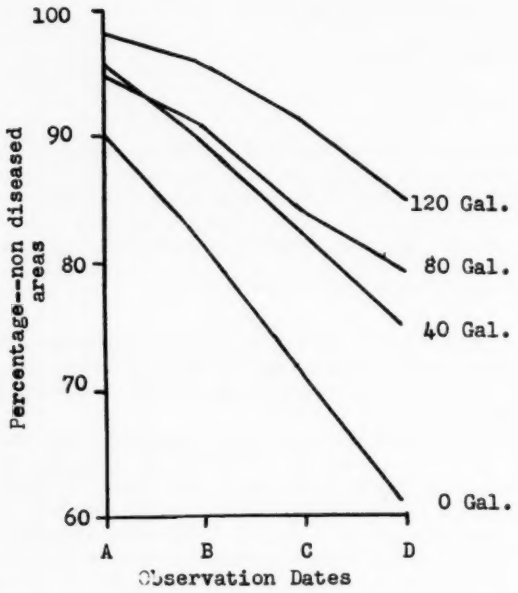

FIGURE 1.
Cumulative areas free from infection after four different fungicidal treatments. Observation dates: (A) July 15, (B) July 30, (C) August 15, (D) August 30.

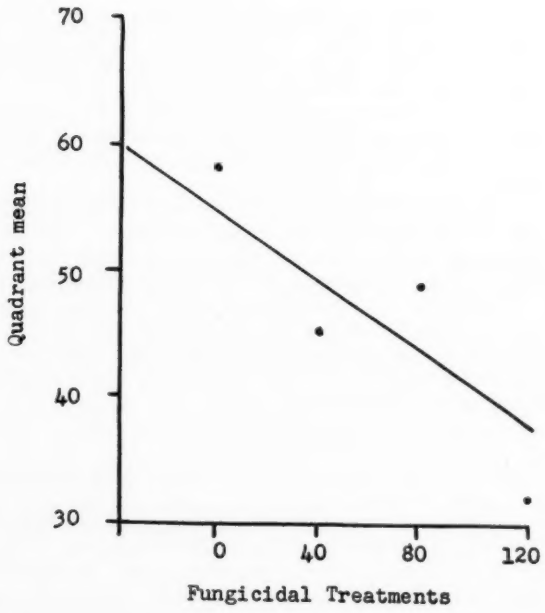

FIGURE 2.
Linear trend for the 0, 40, 80, and 120 gallon per acre fungicidal treatments

by the application of 80 to 120 gallons per acre of CBP-55. This, of course, needs further evaluation.

As a further check on the effectiveness of the fumigant and the method of application, an analysis of variance was made to compare the effects of the last row of the north end and the south end against the remainder of the rows. Calculations for the various sum of squares follow those given by Snedecor (2).

The results of the above analysis can be interpreted as follows: 1) The treatment means were significantly different at the 5 percent level. 2) The investigation demonstrated that the mean quadrants of plants showing symptoms of infection did not deviate significantly at the 5 percent probability level from a linear trend for the 0, 40, 80, and 120 gallon treatments in the experiment. Refer to Figure 2. 3) The end treatments were not significantly different at the 5 percent level from the middle treatments. 4) The interaction of the treatments x ends versus middle were not significantly different at the 5 percent probability level.

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ROOT-KNOT CONTROL IN VEGETABLE CROPS USING D-D AND EDB WITH AND WITHOUT VERMICULITE AS A CARRIER

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Summary

Experimental tests in the field in North Carolina in 1954 and 1955 and demonstration tests in commercial fields and home gardens in 1955 and 1956 showed that root-knot nematode control was as effective in row treatments when vermiculite was used as a carrier for D-D and EDB as comparable liquid treatments. Crops grown in treated soil included beets, broccoli, cabbage, collards, kale, cantaloupe, cucumber, squash, carrots, sweet corn, okra, bean, pole lima bean, pea, pepper, and tomato. In one experimental test D-D treatments appeared to cause a temporary stunting of snapbeans early in the season; however, by time of harvest the stunting effects had disappeared. In one demonstration test beans planted on D-D treated plots did not recover. In all cases the nematocides, irrespective of method of application, gave satisfactory control of root-knot.

Soil application of volatile liquid fumigants on solid carriers is not new. Hammer (3, 4) used vermiculite[1] as a carrier for ethylene chloride and other materials to control the peach tree borer, Synanthedon sp. Taylor and Golden (7) described an effective method of controlling root-knot nematodes using D-D (1, 3-dichloropropene; 1, 2-dichloropropane) and Hi Sil (trade name for a finely powdered silicon compound produced by Columbia-Southern Chemical Company), and pointed out certain advantages of this method of application. In 1954 Sasser and Nusbaum (6) used vermiculite as a carrier for Dowfume W-85 (1, 2-dibromoethane) and obtained satisfactory control of root-knot nematodes on tomato in greenhouse tests and on tobacco in field tests. Nielsen and Sasser (5) in a study of the relationship of nematocides, dosage, carrier and soil types found that fumigants (D-D and Dowfume W-85) carried on vermiculite were more effective at the lower dosages than were the comparable liquid treatments. However, the vermiculite-fumigant mixtures adversely affected yields at the higher dosages, especially in heavy soil.

Investigations reported herein involve field tests in which the two commonly used nematocides, D-D and Dowfume W-85 (EDB), were applied both as liquids as well as mixed with vermiculite for the control of root-knot caused by Meloidogyne incognita acrita Chitwood, 1949. Because of the several advantages of solid application, these tests were designed to compare the relative effectiveness of equal dosages of the two fumigants when applied either on a carrier or as liquids.

MATERIALS AND METHODS

EDB and D-D were added to 60 pounds of vermiculite in tight fiberboard drums at rates of 2 1/4 and 10 gallons of fumigant, respectively. After adding the fumigants the drums were rolled so that the materials would be thoroughly mixed. The drums were left tightly sealed for at least 24 hours prior to use in the field. Vermiculite treatments were applied using various types of fertilizer distributors which made possible the placement of the vermiculite-fumigant mixture in the soil in a band approximately 1 inch wide and at a depth of 6 to 8 inches.

[1] Vermiculite is a lightweight micaceous mineral. A hydrated magnesium-aluminum-iron silicate, vermiculite exfoliates when heated or subjected to certain chemical reactions. It is a monoclinic crystal with pseudo-hexagonal characteristics, with a Mohs scale hardness of 1.5. The material used in these tests was a No. 4 (fine) vermiculite which weighs 10 pounds per cubic foot and the particle size ranges between 30 and 100 mesh. The Zonolite Company, Travelers Rest, South Carolina, generously provided this material.

The mixture was applied at rates of 60 pounds of vermiculite plus the added fumigant per acre so as to give a dosage rate of 2 1/4 and 10 gallons of EDB and DD, respectively. Liquid treatments were applied at the same rates, using gravity flow equipment. Rows were ridged immediately after application. Plantings were made 2 to 3 weeks after treatment. Tests were conducted in 1954 at the Horticultural Crops Research Station, Castle Hayne, North Carolina, and in 1955 at the Vegetable Crops Research Station, Faison, North Carolina. Treatments, which included a vermiculite and/or a non-treated check, consisted of rows approximately 130 feet long and were replicated five times in 1954 and four times in 1955. Yields were taken from 25 feet of row in each treatment. At the conclusion of harvesting a total of 20 plants in 1954 and 10 plants in 1955 were pulled at random from each plot, examined and rated for root-knot severity on the following scale: 1 = no visible galls; 2 = slight; 3 = moderate; 4 = severe; 5 = very severe galling. Root-knot indices are presented as the average of the 10 or 20 plants from each treatment. Crops and varieties tested included: Clemson Spineless okra, Hibiscus esculentus L.; Wade bean, Phaseolus vulgaris L.; and Early Yellow Summer Crooked Neck squash, Cucurbita pepo L.

In 1955 and 1956 these fumigants were evaluated in 35 demonstration tests located in 24 North Carolina counties. These tests were conducted in both commercial fields and in home gardens. Crops grown on soil treated with liquid fumigants and vermiculite fumigant mixtures included: beets, Beta vulgaris L.; broccoli, Brassica oleracea var. italica L.; cabbage, B. oleracea var. capitata L.; collard and kale, B. oleracea var. acephala (L.) DC.; cantaloupe, Cucumis melo L.; cucumber, Cucumis sativus L.; squash; carrots, Daucus carota var. sativa (L.) DC.; sweet corn, Zea mays L.; okra; bean; pole lima bean, Phaseolus lunatus L.; pea, Pisum sativum L.; pepper, Capsicum frutescens L.; and tomato, Lycopersicon esculentum L.

RESULTS

In the 1954 and 1955 tests root knot was not severe early in the season and it was only at the conclusion of harvest that differences in severity of root knot were noted. In 1954 no differences were observed between the root knot ratings of the various treatments on either bean or squash; however, at the conclusion of harvest differences between treatments were obvious on okra (Table 1). While root knot control on okra planted in rows treated with EDB on vermiculite was significantly better than that from the liquid EDB treatment, all chemical treatments were superior to the check treatment. There were no significant differences in yields among treatments on any of the three crops in these tests. However, bean plants grown in soil treated with D-D remained somewhat stunted for a 3-week period after emergence. By harvest time, however, no differences were noted in plant size or yields.

In 1955 root knot ratings were taken on each of the three test crops. In all cases the root knot index of the check plots at the end of the harvest period for each crop showed only moderate infection. There were no significant differences in the severity of root knot between methods of application. Treatments with vermiculite-fumigant mixtures and the liquid fumigants were equally effective in controlling root knot (Table 2). All chemical treatments gave significant control of root knot.

In the 1955 and 1956 demonstration tests, treatments were limited to a comparison of liquid and/or solid applications with a check. Within the 35 tests, the severity of root knot varied from light to very severe. However, in no cases were the vermiculite-fumigant mixtures less effective in the control of root knot than the liquid treatments. With one exception yield increases were obtained through the use of fumigants. At one location beans planted in rows treated with either D-D as a liquid or as a vermiculite mixture were severely stunted and yields were less than those of the non-treated plots.

DISCUSSION

Experimental and demonstration field tests indicate that vermiculite may be used as an effective carrier for D-D and EDB fumigants for root knot control on vegetable crops without decreasing their effectiveness. The use of vermiculite with fumigants offers several advantages over liquids, since it is a free flowing material even after D-D or EDB is added and requires no special application equipment other than a regular fertilizer distributor modified to place the mixture at the proper depth. Hence, methods of calibration for the farmer are essentially the same as those of calibration for the application of fertilizers. While the problem of packaging might be serious, farmers could very easily prepare their own mixtures as

Table 1. The effectiveness of soil fumigant and fumigant-vermiculite mixtures in controlling Meloidogyne incognita acrita on okra in 1954.

Treatment material	Method of application[a]	Root-knot index[b]
EDB	On vermiculite	.616
EDB	Liquid	1.432
D-D	On vermiculite	.758
D-D	Liquid	1.186
Check		3.048
L.S.D. .05		.498
L.S.D. .01		.686

[a]Applied in the row at rates of: D-D, 10 gallons per acre; EDB, 2 1/4 gallons per acre; Vermiculite, 60 pounds per acre.

[b]Root-knot index based on an average of each of 20 plants in each of five replicates using the following scale: 1 = no galls; 2 = slight; 3 = moderate; 4 = severe; and 5 = very severe galling.

Table 2. The effect of soil fumigant and fumigant-vermiculite mixtures on the root-knot indices of okra, bean, and squash in 1955.

Treatment material	Method of application[a]	Average root-knot index[b]		
		Okra	Bean	Squash
EDB	On vermiculite	1.05	1.23	1.35
EDB	Liquid	1.10	1.38	1.28
D-D	On vermiculite	1.35	1.40	1.63
D-D	Liquid	1.20	1.30	1.65
Check	Vermiculite	2.43	2.98	2.95
Check	-	2.33	2.73	3.53
L.S.D. .05		0.52[c]	0.97[d]	1.16[e]
L.S.D. .01		0.72	1.34	1.60

[a]Applied in the row at rates of: D-D, 10 gallons per acre; EDB, 2 1/4 gallons per acre; vermiculite, 60 pounds per acre.

[b]Root knot index based on an average of 10 plants in each of four replicates using the following scale: 1 = no galls; 2 = slight; 3 = moderate; 4 = severe; and 5 = very severe galling.

[c, d, e]Least significant difference for comparing two treatments on okra, beans, squash, respectively.

was done in these tests.

The stunting effect of D-D on bean in two locations confirms previous work (1) and shows that the response of certain crops is an important factor to be considered in soil fumigation.

The use of vermiculite as a carrier for D-D and EDB for the control of root knot in vegetable crops is currently recommended in North Carolina (2).

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DIFFERENTIAL RESPONSE OF HETERODERA SCHACHTII, THE SUGAR-BEET NEMATODE, TO SELECTIONS OF CHENOPODIUM ALBUM

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Summary

Of six selections of Chenopodium album tested for susceptibility to the sugar-beet nematode, one was found to be moderately infected while five were found not to be infected as determined by development of females on the roots. This marked difference in response by the nematode to these selections indicates that there are at least two races of this plant species. Males developed to maturity on five of the selections and were in relatively large numbers on one of these five, suggesting the possibility of further heterogeneity within these selections.

The common weed generally known in the United States as pigweed or lamb's-quarter (Chenopodium album L.) has been reported as a host for the sugar-beet nematode (Heterodera schachtii Schmidt) by various authors, including Corder, Buhrer, and Thorne (1); Filipjev and Schuurmans-Stekhoven (4); Franklin (5); Jones (7); Thorne (14, 15); and Winslow (16). Most of these workers also gave other species of Chenopodium as hosts and Winslow (16) listed three Chenopodium species (C. ambrosioides L., C. botrys L., and C. quinoa Willd.) which he found not be to hosts of this nematode. Jones (7) had also previously reported C. quinoa as not being a host.

McFarlane (8) found in greenhouse tests at Salinas, California that plants of C. album failed to become visibly infected by the sugar-beet nematode obtained locally. Moreover, on several recent occasions the senior author was unable to find by careful examination visibly infected C. album plants growing among sugar beets heavily infected with H. schachtii in the field. These field observations, although subject to many errors, and the findings of McFarlane suggested the possibility that there might be races of this plant species differing in susceptibility to the nematode, that there might be a local race of the nematode to which the plant is highly resistant or even that both possibilities exist.

The present work, preliminary to further investigations along these lines, was conducted to determine if there was a differential response by the local population of the sugar-beet nematode to several selections[2] of C. album[3] obtained from areas in California and Oregon, as specified in Table 1. Since C. album is a variable plant species, these selections were made to represent a range of differences in leaf size and shape and in other characteristics.

MATERIALS AND METHODS

Seeds of sugar beet (Beta vulgaris L.) and of six selections of C. album were germinated in sterilized sand and the resulting plants allowed to grow several days before transplanting. Ten young sugar beet plants and 10 seedlings of each of the selections of C. album were placed individually in aluminum-foil cylinders, 2 1/2 inches in diameter and 7 inches long, which were filled with homogeneously-mixed soil heavily infested with cysts of the sugar-beet nematode. These were grown in the greenhouse for 58 days at a maintained night temperature of 72° F and a variable day temperature of 72° to about 85° F. The plants were then examined for white females and cysts of the sugar-beet nematode and rated individually according to the infection. This was accomplished by examining each plant both macroscopically and microscopically before, during, and after gently washing the roots free of the soil.

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[2]Seeds of these selections were collected originally by Dr. C. W. Bennett, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Salinas, California, and made available by him and one of his assistants, Phyllis Emparan, for testing.

[3]Identified by Dr. S. F. Blake, Plant Taxonomist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

Table 1. Response of the sugar-beet nematode to six selections of Chenopodium album.

Selections	Place and date of original collection of seed	Average infection index[a]		Males (average per pot)
		Plants in cylinders	Plants in pots	
1	Riverside, Calif. 1954	0	0	0
2	Salinas, Calif. 1955	0	0	6
3	Riverside, Calif. 1954	3.3	3.0	21
4	Corvallis, Oregon 1955	0	0	9
5	Corvallis, Oregon 1955	0	0	309
6	Riverside, Calif. 1952	0	0	6
Control (sugar beet)	-------	4.0	---	---

[a]See text for discussion.
Scale: 0 = no infection (no white females or cysts found), 1 = trace (only a few white females or cysts found), 2 = light (white females or cysts in small numbers), 3 = moderate (white females or cysts numerous), 4 = heavy (white females or cysts very numerous).

A Zeiss head-band, binocular magnifier was found very useful in this procedure, although a stereoscopic microscope with higher magnification was used occasionally when considered necessary.

In order to determine if males developed, but possibly without females, two young plants of each of the six selections were placed in each of two 6-inch pots containing heavily infested soil as used in the above test. These plants were grown in the greenhouse with those in the cylinders for the same period of time and under the same environmental conditions. The roots of these plants were then carefully removed from the soil, rinsed lightly in water and examined macro- and microscopically for white females and cysts and rated according to infection, as above. The roots were later macerated in a Waring Blendor (13) and added to 300 grams of thoroughly mixed soil taken from the two pots containing a given plant selection. This soil and chopped root mixture was then processed with slight modification by the sieve and Baermann funnel method (3), after which the recovered H. schachtii males were counted and the total per pot calculated.

RESULTS AND DISCUSSION

Results obtained from these preliminary experiments are presented in Table 1.

Examination of the infection index data shows that only one of the six selections tested became visibly infected by the sugar-beet nematode. Mature females obtained from these roots appeared, upon microscopic examination, to be normal and the cysts were filled with eggs containing living larvae. Of the 10 plants of this susceptible selection tested in cylinders, one had an infection index rating of 2, five had a rating of 3, and four had a rating of 4. Average rating was 3.3, approaching that of the sugar-beet control (4.0). The four plants of this selection grown in pots were also found to be infected, with an average rating of 3.0. All of the plants of selection three, totaling 14, were therefore infected. On the other hand, not a single female was found on the roots of any of the 70 plants of the other five selections. This very marked difference in response of the nematode to these selections is interpreted as evidence that races varying in susceptibility to the sugar-beet nematode exist within this plant species.

The infection index used in the present work indicates the degree of infection as determined by visual estimate of the number of white females and cysts on the roots. The index rating was obtained by scoring each plant individually and then calculating the average for a given selection. This method, adapted with modifications from the relative root-knot index developed by Smith and Taylor (12), is similar to systems used widely in work with root-knot nematodes (2, 10). Mukasa and Ichinohe (9) successfully used the relative root-knot index in studies on the susceptibility of soybean varieties to the soybean-cyst nematode (Heterodera glycines Ichinohe). The modified method as used herein has been found satis-

factory and is relatively quick for indicating the degree of susceptibility of test plants to the sugar-beet nematode. To obtain a more accurate rating, plants should be examined and scored before very many of the females become mature brown cysts, as these are usually more easily dislodged from the roots than are the white females.

Males were found to have developed on five of the selections, although females were observed on the roots of only one of these five. Because of the small number of potted plants involved, owing to a limited supply of seeds, no attempt was made at statistical comparisons of the number of males found in each selection. However, it should be pointed out that the average number of males per pot in selections two, four and six was relatively the same. But the number of males in selection five was about 34 times as great as the number in selection four, and over 50 times as great as the number in selections two and six, a difference of such magnitude as to seem scarcely the result of experimental error entirely. Rather, it would tend to suggest further heterogeneity within this plant species. The development of males on these resistant selections, on which females were not observed, is in line with the findings of Shepherd (11), who reported that about 7 percent of the larvae of the sugar-beet nematode that invaded the roots of the resistant Beta patellaris Moq. developed to maturity as males, and Golden (6) who found that only males of H. schachtii developed to maturity on both B. patellaris and the resistant B. webbiana Moq. No females were found on these Beta species by either worker.

Differences in the development of females of the sugar-beet nematode, as shown by results obtained in the described experiment, indicate that there are two races of Chenopodium album in the selections tested. One is considered a susceptible race, since females apparently developed and reproduced more or less normally, while the other is a highly resistant race on which no females were found to have developed. Moreover, differences in development of males suggest the possibility of further heterogeneity within these selections, since males were found to be in relatively large numbers on one of the selections tested. The existence of such races would provide a plausible explanation for the negative results obtained by McFarlane (8) with this plant and the failure of one of the authors to find infected plants in heavily infested fields, as previously mentioned.

No information has yet been obtained concerning either the occurrence and distribution of the C. album races or the possibility of any cross-breeding of the races which might result in plants having varying degrees of susceptibility, particularly under natural conditions. Further work is being initiated with this plant and certain other plants which might perhaps cause a similar or related response by this parasitic organism. By the use of such plant species, it might be possible in other experiments to determine if there are biologic races of the sugar-beet nematode itself.

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INFLUENCE OF LEAF DIFFUSATE OF SUGAR BEET ON EMERGENCE OF LARVAE FROM CYSTS OF THE SUGAR-BEET NEMATODE (HETERODERA SCHACHTII)

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Summary

Procedures and techniques used in conducting experiments on emergence of larvae from cysts of the sugar-beet nematode are described. Data presented show that larval emergence from cysts in leaf diffusate of sugar beet was more than twice as great as the emergence in plain tap water, but approximately half that from cysts in root diffusate of sugar beet. This indicates that not only the roots but also the leaves of sugar beet give off some substance, or substances, which tend to stimulate emergence of larvae from cysts of the sugar-beet nematode.

The stimulatory effect of root diffusate of sugar beet upon the emergence of larvae from cysts of the sugar-beet nematode (Heterodera schachtii Schmidt, 1871) was demonstrated over 35 years ago. After Baunacke (1) discovered this phenomenon in 1922, Rensch (15) confirmed it in 1924 and various workers later found that diffusate from the roots of a number of other plants, including some which are not hosts, produced a stimulatory effect upon larval emergence. In 1930 O'Brien and Prentice (14) extended this line of work to the golden nematode of potato (H. rostochiensis Wollenweber, 1923) and observed that potato-root diffusate greatly increased emergence of larvae of this species. Since then many other species of Heterodera have been tested for emergence response to root diffusates. As noted by Franklin (7) and shown in further work by Winslow (21), certain of the tested species, notably H. major (Schmidt, 1930) Franklin, 1940, respond only slightly, if any, more in root diffusate than in water. According to Ichinohe (10), larval emergence of the soybean-cyst nematode (H. glycines Ichinohe, 1952) also is not increased when the cysts are placed in root diffusate as compared with water.

Many other materials, including fractional portions of root diffusates, have been examined for possible stimulatory effect on larval emergence of the sugar-beet nematode. For example, Filipjev and Schuurmans-Stekhoven (6) present a review of the earlier work of various investigators, who used without any great success various organic and inorganic materials, including ferric and nitrogenous compounds, animal manures, and whale oil. More recently, Emanuelsson (4) tested a number of vitamins and growth regulators, glutamic and other amino acids, as well as various other organic and miscellaneous substances, including seaweed and pieces of apple. He found that only ascorbic acid increased emergence to any extent; it gave a "hatch" equal to half that obtained from cysts in tap water containing fresh pieces of sugar-beet roots. Thorne (18) obtained a depressing effect on larval emergence with indole-3-acetic acid, theelin, and a root extract prepared from the crushed roots of sugar beet. The depressing effect was evident even when the first two materials were used at very low concentrations. He also observed that vitamin B_1 gave a slight increase in percentage hatch over the water control only at the highest (1:1,000,000) of three concentrations tested.

Wood and Serro (22) found that root diffusate from several plants contained inositol and galactinol, and that the diffusate from two of these, sugar beet and alfalfa, also contained glutamic acid. They further reported that these three chemicals constitute a major portion of the exudate from the roots. In a preliminary "hatching" test, they obtained data suggesting that these three substances produce a stimulatory effect upon emergence of larvae from cysts of the sugar-beet nematode, but drew no conclusions from their results. In further work with galactinol, Swink and Finkner (17) reported an inverse relationship between yield of sugar beets grown on infested land and galactinol content, and suggested that this substance might be used as a chemical characteristic in selecting sugar beets resistant to the sugar-

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beet nematode. Bauserman and Olson (2) separated expressed beet juice into several fractions, and noted that the stimulatory or inhibitory effect of the fractions upon larval emergence from cysts of the sugar-beet nematode may be correlated with certain chemicals present in the fractions. From data obtained in a subsequent larval emergence test, using in pure form 12 of the substances identified in the fractions of expressed beet juice, they tentatively concluded that six of these chemicals (inositol, alanine, isoleucine, gamma amino butyric acid, aspartic acid, and saponin) tended to inhibit larval emergence, while the other six (galactinol, glutamic acid, glycine, valine, glutamine, and sucrose) appeared to increase the emergence of larvae from cysts of the sugar-beet nematode, as compared with distilled water. However, Wallace (20) has shown in extensive, repeated tests with the sugar-beet nematode that there were no significant differences between larval emergence from cysts in water, glactinol, inositol, and glutamic acid, the last three materials being used at several different concentrations. The larval emergence in beet-root diffusate, on the other hand, was significantly higher than in any of these substances. With results tending to support the findings of Wallace (20), Golden (8) has shown in recent tests that emergence of larvae of the sugar-beet nematode from cysts in alfalfa-root diffusate, reported by Wood and Serro (22) in their experiments to have twice as much galactinol as root diffusate of sugar beet, was no greater than emergence in plain tap water. In other experiments Golden (9) found that the root diffusate of Beta patellaris Moq., reported also by Wood and Serro (22) in their tests to contain only about half as much galactinol as root diffusate of sugar beet, produced a very strong effect on larval emergence from cysts of the sugar-beet nematode, an effect similar to that of root diffusate of sugar beet.

Although Massey and Neal (13) reported the discovery of a "hatching agent" in tomato leaves as determined by larval emergence from cysts of H. rostochiensis, there seems to be no published report of such tests for possible effects of leaf diffusate of sugar beet upon emergence of larvae of the sugar-beet nematode. The present work was conducted, after obtaining preliminary results, to determine whether leaf diffusate of sugar beet stimulates emergence of larvae from cysts of H. schachtii, and to gain some idea of its relative effectiveness in comparison with root diffusate of sugar beet and plain tap water.

MATERIALS AND METHODS

Obtaining Treatment Solutions

Root diffusate was collected by leaching tap water through a 4-inch pot containing soil with a vigorously growing sugar-beet plant. Enough water was supplied intermittently during a 24-hour period to produce 200 ml of leachings which passed through filter paper upon dripping from the pot. Leachings were collected at weekly intervals and the same plant was used for 3 successive weeks before being discarded.

Leaf diffusate was obtained by submerging the leaves of vigorously growing sugar-beet plants in 350 ml of tap water for 24 hours. For this purpose each plant was grown in a soil-filled, aluminum-foil cylinder approximately 2 1/2 inches in diameter and 7 inches long. The cylinder was supported horizontally and the leaves bent down under the water in a shallow dish. Since not all the leaves could be submerged at one time, the plants were shifted occasionally and the leaves or parts of leaves not wet were then rinsed with water from the dish. The occasional shifting of the plants also assured better aeration of the submerged leaves. The diffusate was taken in this manner at weekly intervals and after a plant had been used for 3 successive weeks it also was discarded. Care was taken to avoid introducing into the water any soil or debris from the cylinder.

All tap water used was obtained locally from a deep well on the grounds of the United States Agricultural Research Station and was not subjected to any kind of chemical treatment as is usual with city water.

Obtaining Cysts

Young sugar-beet seedlings germinated in sterilized sand were placed in a wooden flat containing soil heavily infested with cysts of the sugar-beet nematode. After growing in this medium for approximately 6 weeks, the plants were transferred to 6-inch pots containing the same type of soil as was used in the flats and grown for about 2 months. Then the plants were removed from the soil, the roots washed, and the wash water and soil screened through 20- and 60-mesh sieves to recover nematode cysts.

The screening procedure was essentially that described by Chitwood (3) for the golden nematode of potato (H. rostochiensis), except that the soil was not dried before processing. All the material from the 60-mesh sieve that did not sink after standing about 30 minutes in the beaker of water was discarded. This eliminated a majority of the old, empty or partially empty cysts considered undesirable for hatching tests, leaving the fresh cysts containing viable eggs and larvae. The remaining contents of the beaker were than poured into Syracuse watch glasses and the cysts were critically examined under a stereoscopic microscope; the ones chosen for experimentation were removed individually be means of forceps and placed in another watch glass containing about 15 ml of tap water. When enough cysts (450) were obtained in this manner for an experiment, they were kept in the refrigerator at approximately 1.1° C for 10 days during which the tap water was drawn off daily with a pipette and replaced with fresh tap water immediately.

Initiating and Conducting the Tests

Immediately after the 10-day period in the refrigerator, the cysts were divided into three uniform cyst groups by selecting with forceps three individual cysts similar in size, shape, color and plumpness and placing one in each of three Syracuse watch glasses containing a small amount of fresh tap water. When each of these watch glasses had received 50 cysts selected in this manner, one watch glass (one replicate) was then assigned to each of the three cyst groups. This whole procedure was then repeated until each cyst group had received three watch glasses (three replicates), with each watch glass containing 50 cysts and giving a total of 450 cysts in one experiment. A second, similar experiment was conducted upon completion of the first one.

Each experiment was conducted for 6 weeks in a dark, aerated cabinet in the laboratory during late summer and fall at a maintained temperature of approximately 25° (day) and 20° C (night). Fifteen ml of fresh treatment solution (tap water, root or leaf diffusate) was placed into each of the nine watch glasses consitituting one experiment. The solutions were changed weekly, at which time the emerged larvae were removed and counted on aliquot when necessary. Nematode counts on individual replicates were not made, but weekly microscopic examination of every replicate indicated that there was very little variation between replicates receiving a given treatment solution. The cysts in each of the three groups composing a given experiment were placed in two different treatment solutions during the 6-week test period. For the first 3 weeks the cysts in cyst groups I, II, and III were in root diffusate, leaf diffusate, and tap water, respectively; during the last 3 weeks of the test, the same cysts in the same group order were in leaf diffusate, root diffusate, and leaf diffusate, respectively. Cysts groups I and II, therefore, received the same treatment solution but in reverse order.

RESULTS AND DISCUSSION

The results obtained from these two experiments are presented in Table 1.

It can be seen from the data in cyst groups I and II of both experiments that there was a rapid emergence of larvae during the first week both in the root and leaf diffusates. Then a progressively smaller number of larvae emerged during each of the 2 succeeding weeks in both of these treatment solutions. In the tap-water control in cyst group III, however, the number of larvae emerging remained comparatively small and varied little each week during this 3-week test period. From the subtotals for root diffusate, it appears that in this treatment solution the number of emerged larvae and the average emerged larvae per cyst were approximately twice as large as in the leaf diffusate. But, on the other hand, the number of larvae emerging in the leaf diffusate was about twice as great as that in the tap-water control, indicating the presence of some substance, or substances, stimulatory to larval emergence in the leaf diffusate.

Further evidence of the stimulating influence of leaf diffusate upon emergence of larvae is shown by the data in cyst group III of both experiments. When leaf diffusate was added to the cysts which had been in tap water the previous 3 weeks, there was a marked increase in emergence with averages somewhat similar to those obtained with this material in cyst group II. However, when leaf diffusate was added to the cysts in cyst group I which had been in root diffusate for the previous 3 weeks and from which a major portion of the hatched larvae had apparently emerged, the progressive decrease in larval emergence continued, as might be expected.

By the use of the procedures described, cysts of relatively uniform size and appearance and of known age, within limits, can be obtained. It would seem that such cysts are less

Table 1. Weekly emergence of larvae of the sugar-beet nematode from cysts given two different treatments during 6-week test period.

CYST GROUPS, TREATMENT SOLUTIONS and TIME of COUNTS	EXPERIMENT[a] 1		EXPERIMENT 2	
	Larvae emerged	Average emerged larvae per cyst	Larvae emerged	Average emerged larvae per cyst
CYST GROUP I -				
Root Diffusate				
1 week	9,852	65.6	14,278	95.1
2 weeks	7,450	49.6	3,464	23.0
3 weeks	742	4.9	1,367	9.1
Subtotal	18,044	120.2	19,109	127.2
Leaf Diffusate				
4 weeks	533	3.4	616	4.1
5 weeks	115	0.7	122	0.8
6 weeks	78	0.5	17	0.1
Subtotal	726	4.8	755	5.0
Total	18,770	125.0	19,864	132.2
CYST GROUP II -				
Leaf Diffusate				
1 week	4,631	30.8	4,226	28.1
2 weeks	2,695	17.9	2,262	15.0
3 weeks	1,285	8.6	2,247	14.9
Subtotal	8,611	57.3	8,735	58.0
Root Diffusate				
4 weeks	9,408	62.7	10,783	71.8
5 weeks	2,087	13.9	2,075	13.8
6 weeks	156	1.0	397	2.6
Subtotal	11,651	75.6	13,255	88.2
Total	20,262	133.0	21,990	146.2
CYST GROUP III -				
Tap Water (Control)				
1 week	1,448	9.6	1,459	9.7
2 weeks	1,550	10.3	1,107	7.3
3 weeks	1,246	8.3	782	5.2
Subtotal	4,244	28.2	3,348	22.2
Leaf Diffusate				
4 weeks	5,186	34.5	3,370	22.4
5 weeks	3,701	24.6	1,585	10.5
6 weeks	588	3.9	360	2.4
Subtotal	9,473	63.1	5,315	35.3
Total	13,717	91.3	8,663	57.5
Grand Total	52,749	116.4	50,517	111.9

[a]Each of the two experiments consists of 450 cysts equally divided into three cyst groups with each group composed of 3 replicates of 50 cysts each.

variable than those collected by methods which involve excessive drying of the soil before extracting the cysts or by using all cysts from the soil without critical selection. As Wallace (20) pointed out, the cysts should be kept moist because it has been shown previously by Wallace (19) that drying of sugar-beet nematode cysts before initiation of "hatching" tests reduced the subsequent larval emergence. The value of cyst selection is indicated by the reasonably uniform and consistent results obtained in both experiments, especially in cyst groups I and II in which the treatments were the same for both groups but were applied in reverse order. Another factor possibly contributing favorably to the results obtained was the 10-day period of refrigeration of the cysts. This treatment was given for three main reasons: 1) to give the cysts a water pre-soak before initiation of the test, such as suggested by Fenwick (5) for H. rostochiensis, and at the same time prevent larval hatching and emergence as well as the growth of contaminating microorganisms; 2) to attempt to remove by the daily changes of water any "hatching factor" which might be present upon or within some of the cysts; and 3) to reduce to a very low state the metabolic activities of all larvae and developing eggs within the cysts.

From the results of these tests it appears that not only the roots but also the leaves of sugar beet produce or give off a substance, or substances, which stimulates emergence of larvae from cysts of the sugar-beet nematode. As pointed out by Kramer (11), it has been demonstrated that plants lose salts and organic materials by leaching of the foliage with water. For example, Long, Sweet and Tukey (12) found by analysis of foliage "leachate" (diffusate) that bean plants (Phaseolus vulgaris L.) lost a large percentage of potassium (K^{42}) and smaller amounts of rubidium (Rb^{86}), the loss being greatest in plants which had been both in the dark during the nutrient absorption period and in the medium of high initial salt content. The diffusate was also found to contain "four or five" amino acids and large quantities of galactan. Chemical analysis of the leached plants further showed approximately a 10 percent reduction in the content of calcium, potassium, magnesium, nitrogen, and phosphorus. More recently, Sprent and Barber (16) reported leaching from young seedlings of garden peas (Pisum sativum L.) with water, a "flower-inhibitor", an unidentified substance delaying flowering in garden peas. But whether the stimulatory material apparently present in leaf diffusate of sugar beet is related to the substances already identified or found in leaf diffusates from other plants is not known, as no data have been obtained on its chemical and physical properties.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, SALINAS, CALIFORNIA

NEW HOST RECORDS FOR HETERODERA GLYCINES; INCLUDING ONE HOST IN THE LABIATAE

James M. Epps[1] and A. Y. Chambers[2]

Summary

The host range of the soybean cyst nematode, Heterodera glycines, is extended as a result of a series of tests in the greenhouse. The new hosts reported include hemp sesbania (Sesbania macrocarpa); white lupine (Lupinus albus), and henbit deadnettle (Lamium amplexicaule). The last-named is a member of the Labiatae and is the first host reported outside the Leguminosae.

Since the discovery of the soybean cyst nematode, Heterodera glycines Ichinohe, 1952, in Tennessee in 1956, tests have been conducted in the greenhouse to find additional host plants for this species. Plants previously reported (1, 2, 3) as hosts include soybean, Glycine max (L.) Merr.; wild soybean, Glycine ussuriensis Regel & Maack; snapbean, Phaseolus vulgaris L.; common vetch, Vicia sativa L.; Korean lespedeza, Lespedeza stipulacea Maxim.; and adsuki bean, Phaseolus angularis Wight. (The adsuki bean is a food bean grown in the Orient.)

In a series of plantings which included cultivated as well as wild plants three new hosts were discovered. These were hemp sesbania, Sesbania macrocarpa Muhl.; white lupine, Lupinus albus; and henbit deadnettle, Lamium amplexicaule L. The last-named belongs to the Labiatae and is the only known host outside the Leguminosae.

Soil from a field known to be heavily infested with H. glycines cysts was brought into the greenhouse and placed in 6-inch clay pots. Various kinds of cultivated and wild plants were planted in the pots. Approximately 6 weeks after planting the plants were removed from the soil, washed, and examined under the dissecting microscope for the presence of white females. If white females were present they were placed in a Syracuse watch glass to determine if active second-stage larvae would emerge. Emergence of active larvae indicated that the full life cycle had been completed on the above hosts. These tests were repeated. Additional tests are being conducted.

Hemp sesbania is a common weed in fields, especially in the low lands along the Mississippi River, and is known locally as coffee bean. Henbit deadnettle is a winter annual found growing in waste and cultivated land. White lupine is grown as a winter crop in the South.

From the tests it appears that although the soybean cyst nematode can complete its life cycle on these plants, it produces fewer white females on them than on soybeans grown under the same conditions, which indicates that these plants may not be preferred hosts for the nematode.

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COOPERATIVE INVESTIGATIONS OF THE CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND THE TENNESSEE AGRICULTURAL EXPERIMENT STATION

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NEMATODES ASSOCIATED WITH MINNESOTA CROPS. I. PRELIMINARY SURVEY OF NEMATODES ASSOCIATED WITH ALFALFA, FLAX, PEAS, AND SOYBEANS[1]

Donald P. Taylor, Roger V. Anderson, and William A. Haglund[2]

Abstract

Nematodes were identified from 40, 30, 56, and 90 soil samples from fields of alfalfa, flax, peas, and soybeans, respectively, in Minnesota. Most commonly identified were species of the genera Xiphinema, Tylenchorhynchus, Helicotylenchus, Paratylenchus, and Pratylenchus. In addition nematodes of the genera Aphelenchoides, Aphelenchus, Criconemoides, Ditylenchus, Heterodera, Hoplolaimus, Meloidogyne, Boleodorus, Neotylenchus, Nothotylenchus, Psilenchus, Rotylenchus, Trichodorus, and Tylenchus, were found.

In 1956 the Minnesota Agricultural Experiment Station initiated a study of nematodes, one objective of which was to identify plant parasitic nematodes associated with Minnesota crops. Prior to that date only the genera Aphelenchoides Fischer, 1894 (6), Heterodera Schmidt, 1871 (7), and Meloidogyne Goeldi, 1887 (6) had been reported from Minnesota. Recent surveys in several other States have shown that plant parasitic nematodes are common in agricultural soils in all regions of the United States where an attempt has been made to find them (3, 4, 5, 8). It seemed probable that they might also be common in agricultural soils in Minnesota, and the survey here described was undertaken to determine this.

During the latter part of the 1956 growing season and throughout 1957, 216 soil samples were collected from Minnesota alfalfa, flax, pea, and soybean fields by the writers and other members of the Department of Plant Pathology and Botany. Samples were taken at random throughout the State without any effort to obtain uniform geographical coverage. The number of samples from each of the 58 counties where samples were taken are listed in Table 1.

Each sample was a composite of 10 to 20 subsamples taken from scattered sites within a field. Each subsample consisted of a core of soil 1 inch in diameter and 8 to 12 inches long taken from the rhizosphere of actively growing plants. Samples were processed by a combination screening and funnel technique, similar to Christie and Perry's method (2). The fractions containing nematodes were collected on #80 and #270 U. S. Standard Testing Sieves.

Temporary mounts of stylet-bearing nematodes were made by placing heat-relaxed specimens in 3% formaldehyde on a microscope slide. Identifications were made using a compound microscope with an oil immersion objective.

Data on the occurrence of known or suspected plant parasitic nematodes are summarized in Table 2. The most frequently encountered genera, believed to contain only obligate plant parasites, were Xiphenema Cobb, 1913, Tylenchorhynchus Cobb, 1913, Helicotylenchus Steiner, 1945, Paratylenchus Micoletzky, 1922, and Pratylenchus Filipjev, 1934.

Xiphinema americanum Cobb, 1913 was the most frequently recovered species, occurring in about 75 percent of the soil samples collected from alfalfa, flax, and soybean fields. It was found in less than half of the pea fields examined.

Species of Tylenchorhynchus, found in 52 percent of the samples, were the next most frequently identified forms. Members of this genus were particularly common in soil samples from flax fields.

Helicotylenchus spp. and Paratylenchus spp. each occurred in 45 percent of the 216 samples. Helicotylenchus spp. were slightly less common in soil from alfalfa fields than in soil samples from the other crops; Paratylenchus spp. were more constantly associated with the four crops.

Species of Pratylenchus, root lesion nematodes, occurred in 41 percent of all samples collected and in 77 percent of the samples from flax fields.

Two other species are believed to be of importance although they occurred less frequently

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Table 1. Distribution by county of soil samples from alfalfa, flax, peas, and soybeans.

County	A.[a]	F.[b]	P.[c]	S.[d]	T.[e]	County	A.[a]	F.[b]	P.[c]	S.[d]	T.[e]
Aitkin	1	-	-	-	1	Mahnomem	-	1	-	-	1
Anoka	1	-	-	3	4	Martin	-	1	-	1	2
Becker	-	1	-	-	1	Meeker	-	-	-	2	2
Beltrami	1	-	-	-	1	Mille Lacs	-	-	-	3	3
Benton	-	-	-	1	1	Mower	-	-	6	4	10
Big Stone	-	1	-	-	1	Nobles	-	1	-	-	1
Blue Earth	1	-	-	4	5	Norman	-	1	-	-	1
Brown	1	-	-	4	5	Olmsted	1	-	-	3	4
Carver	5	-	-	2	7	Pennington	-	1	-	-	1
Chippewa	-	1	-	4	5	Pipestone	-	-	-	1	1
Chisago	-	-	-	5	5	Polk	1	1	-	1	3
Clay	1	-	-	2	3	Pope	1	-	-	-	1
Cottonwood	1	5	-	2	8	Ramsey	1	1	-	-	2
Dakota	5	2	3	4	14	Redwood	1	2	-	2	5
Dodge	-	-	-	1	1	Renville	1	1	1	1	4
Faribault	-	-	7	3	10	Rice	-	-	1	-	1
Fillmore	-	-	10	2	12	Rock	1	-	-	-	1
Freeborn	2	-	-	2	4	St. Louis	1	1	4	-	6
Goodhue	1	-	9	4	14	Scott	2	-	2	-	4
Isanti	-	-	-	4	4	Sherburne	-	-	-	4	4
Itasca	1	-	-	-	1	Sibley	1	-	4	7	12
Kanabec	1	-	-	3	4	Stearns	-	-	-	1	1
Kandiyohi	-	-	-	1	1	Swift	-	1	-	-	1
Koochiching	1	-	-	-	1	Traverse	-	1	-	-	1
Lac qui Parle	-	1	-	-	1	Wabasha	-	-	1	-	1
LeSueur	-	-	7	-	7	Wadena	1	-	-	-	1
Lincoln	-	2	-	-	2	Washington	2	-	-	2	4
Lyon	-	2	-	-	2	Wilkin	-	1	-	1	2
McLeod	1	-	1	2	4	Yellow Medicine	2	1	-	4	7
Total							40	30	56	90	216

[a] Alfalfa
[b] Flax
[c] Pea
[d] Soybean
[e] Total samples per county

Table 2. Nematodes associated with alfalfa, flax, peas, and soybeans in Minnesota.

Nematode species	Crop Plant									
	Alfalfa 40 Samples		Flax 30 Samples		Pea 56 Samples		Soybean 90 Samples		Total 216 Samples	
	n[a]	p[b]	n	p	n	p	n	p	n	p
Aphelenchoides spp.	3	8	4	13	3	5	8	9	18	8
Aphelenchus spp.	29	73	28	93	46	82	59	66	162	75
Criconemoides rusticum	1	3	-	-	-	-	-	-	1	1
Ditylenchus spp.	10	25	6	20	5	9	22	24	43	20
Helicotylenchus spp.	-	-	3	10	-	-	3	3	6	3
H. erythrinae[c]	7	18	10	33	26	46	39	43	82	38
H. nannus	7	18	1	3	-	-	7	8	15	7
Total	12	30	13	43	26	46	47	52	98	45
Heterodera spp.	1	3	4	13	-	-	1	1	6	3
H. cacti-group	2	5	-	-	-	-	6	7	8	4
H. trifolii	-	-	-	-	2	4	-	-	2	1
Total	3	8	4	13	2	4	7	8	16	7
Hoplolaimus coronatus	9	23	19	63	12	21	23	26	63	29
Meloidogyne spp.	1	3	1	3	-	-	-	-	2	1
Neotylenchidae										
Boleodorus spp.	6	15	-	-	-	-	11	12	17	8
Neotylenchus spp.	3	8	2	7	1	2	8	9	14	6
Nothotylenchus spp.	7	18	1	3	10	18	18	20	36	17
Paratylenchus spp.	17	43	14	47	32	57	35	39	98	45
Pratylenchus spp.	1	3	5	17	2	4	1	1	9	4
P. hexincisus[d]	5	13	10	33	9	16	10	11	34	16
P. minyus	1	3	-	-	6	11	-	-	7	3
P. penetrans	8	20	7	23	-	-	26	29	41	19
P. pratensis	1	3	1	3	-	-	8	9	10	5
P. scribneri	1	3	1	3	-	-	1	1	3	1
Total	13	33	23	77	16	29	37	41	89	41
Psilenchus spp.	6	15	6	20	5	9	24	27	41	19
Rotylenchus spp.	-	-	1	3	-	-	-	-	1	1
R. robustus	-	-	2	7	-	-	1	1	3	1
Total	-	-	3	10	-	-	1	1	4	2
Trichodorus spp.	1	3	-	-	9	16	15	17	25	12
Tylenchorhynchus spp.	2	5	3	10	-	-	6	7	11	5
T. acutus	2	5	7	23	1	2	3	3	13	6
T. brevidens	-	-	-	-	1	2	-	-	1	1
T. clarus	-	-	-	-	4	7	-	-	4	2
T. dubius	-	-	-	-	4	7	-	-	4	2
T. latus	5	13	5	17	10	18	10	11	30	14
T. leptus	1	3	-	-	-	-	-	-	1	1
T. maximus	1	3	-	-	-	-	2	2	3	1
T. nudus	9	23	10	33	9	16	30	33	58	27
T. striatus	-	-	-	-	2	4	1	1	3	1
Total	17	43	21	70	28	50	47	52	113	52
Tylenchus spp.	31	78	23	77	46	82	64	71	164	76
Xiphinema americanum	30	75	22	73	23	41	70	78	145	67

[a]Number of samples in which each form occurred.
[b]Percentage of samples in which each form occurred.
[c]Helicotylenchus erythrinae - includes both this species and a closely related undescribed species.
[d]Pratylenchus hexincisus - includes all forms having six incisures in the lateral field.

than the five genera mentioned above. Hoplolaimus coronatus Cobb, 1923 occurred in 63 percent of the samples from flax fields although its over-all frequency of occurrence was 29 percent. An unidentified species of Trichodorus Cobb, 1913 was found in large numbers and in a high percentage of soil samples from soybean fields from a rather restricted area north of St. Paul.

All nematode species listed in Table 2, except the Heterodera cacti group, are first reports of the species from Minnesota, indicating that they have a wider geographical distribution than had previously been reported. For example, Tylenchorhynchus acutus Allen, 1955, occurred in 6 percent of the samples, yet prior to this paper only three specimens of this species had been reported (1).

In many of the samples populations of plant parasitic species were large, indicating that nematode damage to crops probably occurs commonly in Minnesota. Tests are in progress to determine the host ranges of some of the more commonly occurring species, and pathogenicity to several Minnesota crops. Future surveys are planned to determine the distribution of plant parasitic nematodes on additional crops in Minnesota.

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A NOTE ON NEMATODES ATTACKING COFFEE TREES IN BRAZIL

Luiz Gonzaga E. Lordello and Adiel Paes Leme Zamith

The first record on nematode parasites of coffee tree roots in Brazil was published by Jobert (8) in 1879. Some years later, Goeldi (2) stated that the primary cause of a disease affecting coffee plantations at the so called Province of Rio de Janeiro was the nematode referred to by Jobert. At that time, Goeldi erected the new genus Meloidogyne, and described M. exigua as the type species.

Since then, authors have made references to M. exigua found infecting roots of coffee trees in several South American countries and in the U.S.A., at the New York Botanical Garden (1, 4, 5).

Recently, the writers examined roots of coffee plants from the Ribeirão Prêto area, in the state of São Paulo, and found M. exigua attacking all the decaying trees. Experiments for recuperating old coffee orchards have been conducted at one of the plantations infected by M. exigua, but the trees did not give the expected responses.

On the roots of coffee trees M. exigua produces somewhat small galls which may be easily overlooked, particularly if the material collected is not protected against desiccation. Necrotic areas are also to be seen on the roots. (Fig. 1).

Preliminary surveys suggest that M. exigua is an important detriment to coffee production in the Ribeirão Prêto region.

The trees examined belong to the following varieties of Coffea arabica L.: red Bourbon, yellow Bourbon, and Mundo Novo.

FIGURE 1. Roots of coffee trees attacked by Meloidogyne exigua Goeldi, 1887.

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SOME VIRUS-LIKE DISORDERS OF POME FRUIT TREES IN MISSOURI[1]

A. F. Posnette and D. F. Millikan

Abstract

Five previously unreported disorders on pome fruits are recorded. These disorders and the virus disease they resemble are chat fruit, rough skin and rubbery wood on apple and vein yellows and red mottle on pears. The occurrence of these disorders in commercial orchards suggests that the need for precautions in propagation is no less for pome than for Prunus fruit trees, for which scion certification programs are already in operation.

Many virus diseases are known to occur in pome fruit trees in Europe, but few have been reported in North America. A brief survey of orchards in Missouri has indicated that this implied difference may be more apparent than real. Prior to this survey only one virus disease of apple, stem pitting, was known in Missouri, but symptoms resembling those of several virus diseases not previously recorded in America have now been found. Although the identity of these diseases has not yet been proved by transmission tests, the proximity of normal and affected trees in orchards supported the diagnoses based on symptomatology.

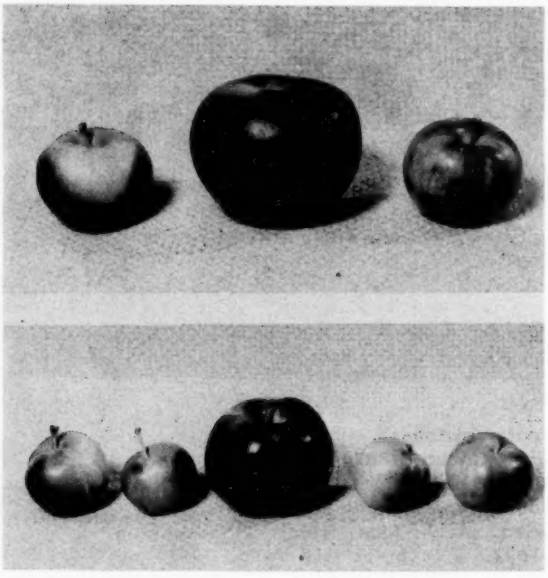

FIGURE 1. Reduced fruit size and poor coloration on Turley Winesap. Left--"chat fruit" condition; center--healthy; right--unidentified mottle condition thought to be similar to dapple apple.

FIGURE 2. Reduced fruit size, distorted shape, and stem end scarring on Jonathan affected with "rough skin". Center--healthy control.

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The diseases of which suspected symptoms were seen are listed below.

Fruit	Disease	Varieties Affected	References
Apple	Chat fruit (Fig. 1)	Jonathan, Turley, Delicious	Luckwill & Crowdy (4)
	Rough skin (Fig. 2)	Golden Delicious, Jonathan	van Katwyk (2)
	Rubbery wood	Golden Delicious, Stayman	Prentice (6)
Pear	Vein yellows	Oregon 63, others	Posnette (5)
	Red mottle	Pyrus calleryana	Posnette (5)

Other observed disorders that could be attributed to virus diseases already reported in North America include stony pit of pear (3) and apple mosaic (1). One other disorder resembling dapple apple (7) was also found for the first time in Missouri (Fig. 1).

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TWO STONE FRUIT VIRUS DISEASES NOT PREVIOUSLY REPORTED FROM WEST VIRGINIA[1]

Robert E. Adams[2]

Two stone fruit virus diseases were observed during the growing season of 1957 that have not previously been reported from West Virginia. Prune dwarf was found on Italian prune (Prunus domestica L.) and what is considered to be peach mosaic was observed on Elberta peach (Prunus persica (L.) Batsch) and on a peach of unknown variety.

The discovery of prune dwarf in a small planting of prunes in the vicinity of Romney, West Virginia was not surprising because sour cherry yellows, sometimes associated with prune dwarf[3] is prevalent in all sour cherry plantings in this State. Since prune culture in West Virginia is limited to small-scale production for home use and a few small commercial plantings, this report is of little economic interest but it extends the known geographical range of the disease.

Peach mosaic is a destructive disease in the Southwestern and Western States (1). Its occurrence in any commercial peach growing section is cause for concern.

Peach fruits having an appearance practically identical with the published pictures and descriptions of peach mosaic-affected fruits were found in two commercial orchards in Hampshire County. They were strikingly similar to the pictures on plate 4, U.S.D.A. Handbook 10[3].

The first discovery was in an Elberta planting near Three Churches, West Virginia, where two trees bearing typical bumpy fruits were found in late summer. One tree was systemically infected, with diseased fruits occurring throughout. The other tree showed symptomatic fruits on only one scaffold branch. Close examination of the foliage on diseased current-season growth revealed a slight mottle on the first formed leaves. Leaves produced in mid-season were symptomless but those formed in late season with the advent of cool weather showed the mottle. In many instances, the mottled portions then observed were being cut out and dropped from the leaf. Terminal growth on the diseased trees was less than that on adjoining trees, and terminal growth of the current season was less than that made by the same trees during 1956. The diseased fruits were of normal flavor and texture but ripened earlier than normal fruits.

The grower reports that this season is the first in which abnormal fruits have been produced. This orchard has been visited and examined periodically every growing season since 1953 and the condition has not been noted previously. This fact, together with the sectoral appearance of the disease on one tree, suggests that these trees became infected late in 1956 or early in 1957.

Most of the trees of an unknown early variety in an orchard near Romney also bore fruits symptomatic of peach mosaic. These trees seem to have been affected for several years and they have been injured to such an extent that the grower considers the variety worthless. No other peach trees in the vicinity of this planting were found to be affected. However, many of the trees of other varieties in the immediate vicinity of the diseased planting had been harvested before the disease was found.

It has been recommended that all the diseased trees be destroyed this fall in an attempt to eradicate the disease. Surveys for the prevalence of peach mosaic should be made during the coming growing season to determine whether these infections are isolated cases.

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[3]Anon. Virus diseases and other disorders with virus like symptoms of stone fruits in North America. United States Department of Agriculture. Agriculture Handbook 10, 1951.

AN APPARENTLY NEW VIRUS DISEASE OF PEACH[1]

Robert E. Adams[2]

Abstract

A disease of suspected virus etiology has been found in a planting of Freeland peaches at Kearneysville, West Virginia. The most striking symptoms are delayed foliation in the spring, decline, abortion of the majority of the developing embryos, failure of fruits with aborted embryos to develop, and copious gum exudation from trunks, scaffold branches, and fruits. Transmission studies indicate that necrotic ringspot virus, peach necrotic leafspot virus, and an unidentified virus are present in diseased trees and the disease is tentatively designated embryonic abortion.

INTRODUCTION

In the spring of 1957 intensive observations and studies were begun in a planting of Freeland peaches (Prunus persica (L.) Batsch) near Kearneysville, West Virginia. Despite the relatively young age (9 years) of this planting it has shown decline, poor yield of marketable fruit, and severe brown rot incidence for several years.

The purpose of this paper is to report the occurrence in West Virginia of an apparently new peach disease responsible for the decline in this orchard, to describe briefly the symptoms observed, and to present evidence that it is transmissible by grafting.

SYMPTOM COMPLEX ON NATURALLY INFECTED TREES

Symptoms are transitory on foliage. Foliage appears on diseased trees about 1 week later than normal and early terminal growth is rosetted. There is a slight mottle on the first leaves. As the temperature rises with the advancing season, the mottle fades and the trees grow out of the rosetted condition. This is followed by foliage symptoms which appear to be the same as those described for peach necrotic leafspot (1).

There is a heavy set of blossoms. However, the majority of the embryos abort shortly after the fruits begin to develop. Fruits with aborted embryos fail to develop but hang on the trees all season (Figure 1). Gum pockets develop within most of them and they exude gum profusely. The few fruits with normal embryos attain normal size but 30 to 40 percent of them contain gum pockets within the flesh and they also show pronounced gummosis (Figure 2). Diseased fruits are extremely susceptible to infection by Monilinia fructicola (Wint.) Honey. Gum pockets which exude gum profusely are also formed in abundance on the scaffold branches and trunk (Figure 3). Microscopic examination of freehand sections indicates that gum formation is associated with the collapse of the phloem tissue. The xylem was rarely involved. In a few instances gum pockets included within the xylem were observed in stems. Dieback is common and one or more of the main scaffold branches is usually dead and others show symptoms of decline.

INOCULATION STUDIES

Culturing and microscopic examination of diseased tissues have failed to reveal any bacteria or fungi believed to be capable of causing this condition. No evidence of insect attack has been found that could have been responsible. Bud indexing of diseased trees to peach seedlings and Shirofugen trees and approach grafting of healthy trees, growing in pots, to diseased orchard trees produced the symptoms described below on five hosts.

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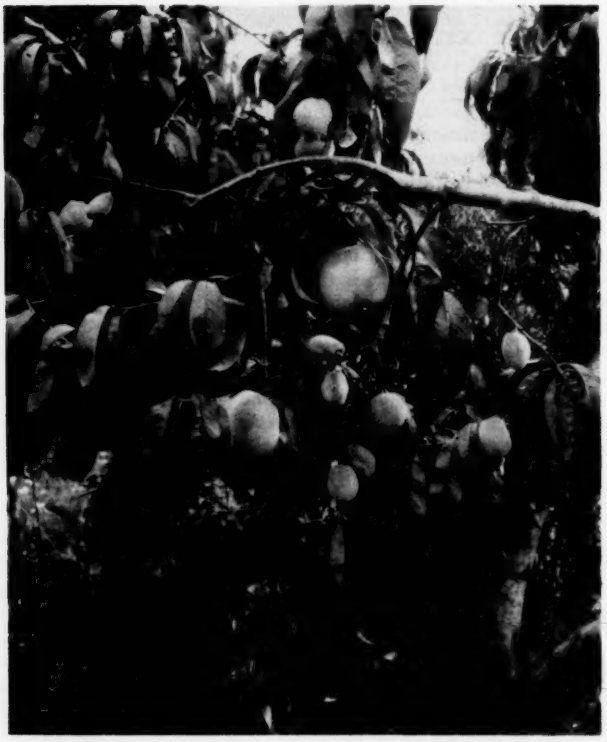

FIGURE 1. Freeland peach tree affected with embryonic abortion, showing normal fruits, and small fruits (buttons) with aborted embryos. (Photograph by H. H. Lyon, Jr., Department of Plant Pathology, Cornell University, Ithaca, New York.)

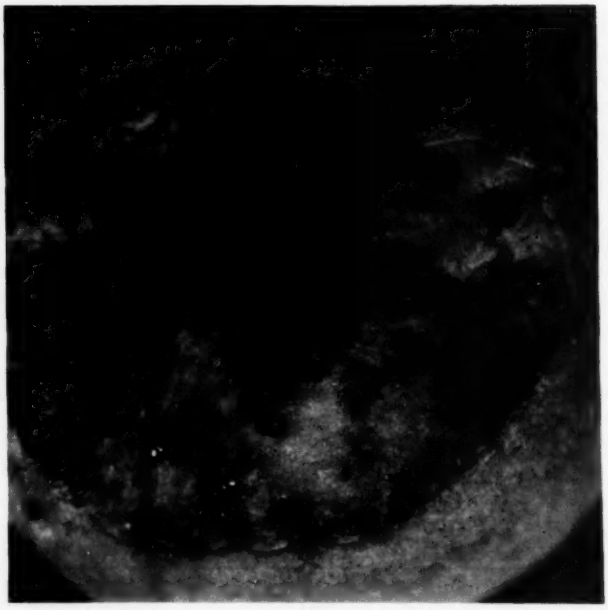

FIGURE 2. Freeland peach exhibiting symptoms of embryonic abortion, cut to show the character of the gum pockets produced and their internal distribution in diseased fruits. (Photograph by H. H. Lyon, Jr., Department of Plant Pathology, Cornell University, Ithaca, New York.)

FIGURE 3. Scaffold branch of Freeland peach with embryonic abortion, showing the characteristic gum exudation from affected trunks and scaffold branches. (Photograph by H. H. Lyon, Jr., Department of Plant Pathology, Cornell University, Ithaca, New York.)

On Shirofugen: Prunus serrulata Lindl. var. sachalinensis form albo-rosea (P. pseudo-cerasus var. Shirofugen Spaeth).

Diseased trees, when indexed to Shirofugen, produced a reaction characteristic of necrotic ringspot. This indexing and symptom reading was done by Dr. Fred Lewis, Plant Pathologist, Pennsylvania State University, Fruit Research Laboratory, Arendtsville, Pennsylvania. The interest and active cooperation of Dr. Lewis in this undertaking are gratefully acknowledged.

On Mazzard Seedlings: Prunus avium L.

Actively growing potted seedlings were approach-grafted to diseased peach trees. Large diffuse chlorotic spots were produced on the leaves of the seedlings which developed shortly after the approach grafts were made. The etch type symptoms of necrotic ringspot appeared on leaves that developed later.

On Mahaleb seedlings: Prunus mahaleb L.

Leaves produced on Mahaleb seedlings shortly after being approach-grafted to diseased trees showed a pronounced vein clearing. This was followed by apparent recovery as indicated by the lack of symptoms on leaves produced later. The vein clearing symptom on foliage was similar to that described as golden-net on apricot, plum, and peach (1).

On Montmorency budlings: Prunus cerasus L.

The symptom produced on Montmorency foliage subsequent to approach grafting was a diffuse, broad, vein banding chlorosis.

On Elberta seedlings: Prunus persica (L.) Batsch.

The initial symptom on approach-grafted seedlings was a slight mottle that soon faded. This was followed by symptoms of peach necrotic leafspot. These symptoms were the same as those observed on diseased orchard trees.

Seedlings, bud-inoculated in the field, produced gum profusely and immediately at the site of inoculation. The appearance of foliage symptoms on these seedlings will of necessity be delayed until spring growth in 1958.

There has been no opportunity to learn whether fruit symptoms are transmissible.

DISCUSSION

It is likely that necrotic ringspot is a part of this disease complex with the added possibility that peach necrotic leafspot and an unknown virus are involved. It is not known whether these three viruses in combination will produce the symptom complex observed in the field or whether there is still another virus in the complex.

It is possible that this disease may reduce a tree's tolerance to low winter temperatures and that winter injury may be responsible for some of the symptoms observed, such as dieback.

This disease has been found on only one farm in West Virginia but because of its lethal character, and apparent rapid spread within the orchard, attempts are being made to eradicate it by removal and destruction of known diseased trees. Circumstantial evidence leads to the conclusion that the disease was introduced in the planting stock. Since the Freeland variety has not been released for general planting by the nursery trade, there may be a possibility of eliminating the only known source of inoculum before the disease becomes a serious threat to the peach growing industry.

This disease is tentatively designated embryonic abortion.

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PIERCE'S DISEASE VIRUS IN MISSISSIPPI AND OTHER SOUTHERN STATES

Wm. B. Hewitt[1], N. H. Loomis[2], J. P. Overcash[3], and G. K. Parris[4]

Evidence that the virus which causes Pierce's disease of grapevines occurs in States other than California became evident in 1942 when the disease was identified by Hewitt in grape specimens from Carrizo Springs, Texas (5). Reports of the disease in grapevines in Florida (7) indicated that the virus might occur in other Gulf States. An examination of some experiment station records of Southern States showed that European and many of the American bunch grapes would not survive in plantings in some of the Southern States (6). Though many plants were reported to grow vigorously the first season or so, many of them would live from only 2 to perhaps 4 years after planting. These reports also indicate that some varieties live much longer than others. Furthermore, history relative to the first stages of the development of the disease in California indicated that the virus was introduced into the State possibly in grape cuttings or rootings from the Mississippi Valley (6). To determine the possible presence of the Pierce's disease virus in another southern State, tests were set up at State College and at Meridian, Mississippi.

METHODS

Rootings of Emperor grape, a variety of Vitis vinifera which will show very good symptoms of Pierce's disease, were grown in the nursery at Davis, California. The plants were dug in the fall and lots selected at random. One lot was sent to State College and another to Meridian, Mississippi, where they were planted out in local experimental field plots. A third lot of vines, also selected at random from the same batch of rootings, was planted in nursery cans and grown in the glass house at Davis for control plants.

At State College the plants were set out in the horticulture field plantings, and at Meridian they were planted in the vineyard at the United States Horticultural Field Station. All plants were grown for one season in their respective plantings. In the succeeding fall some of the surviving plants at State College and at Meridian were dug and returned to Davis, where they were planted in nursery cans and placed in the glass house along with the control plants. These plants were observed for symptoms of Pierce's disease at intervals throughout the growing season subsequent to their return to Davis. The presence of Pierce's disease virus in plants was determined first by symptoms, then by graft transmission to healthy grape plants, and also by insect vector transfer to grape and alfalfa.

EXPERIMENTAL TESTS

In December 1951 lots of 20 vines each were sent to State College and Meridian where they were planted. Ten vines were grown in cans in the glass house at Davis. By December 1952 only six of the plants at State College were living. Three of these were dug and returned to Davis. Nine plants were living at Meridian and seven of them were returned to Davis. The 10 plants transferred from Mississippi were planted in nursery cans and held along with the 10 control plants in the glass house at Davis. During the spring of 1953 only two of the three plants from State College and only three of the seven plants from Meridian leafed-out and grew. All 10 control plants grew. None of the 15 grape plants showed symptoms of Pierce's disease during 1952 or 1953.

In a second test in December 1952 a lot of 15 healthy Emperor rootings was sent to State College, and a lot of 25 Emperor rootings was sent to Meridian, where they were planted out in field plots. Ten plants were grown in the glass house at Davis as controls. In the fall of 1953 there were only a few plants remaining at State College and these survivors were in various stages of degeneration. None of the plants were returned to Davis. Six Emperor plants were returned to Davis in December, 1953 from Meridian. They all started growth in the glass house at Davis in the spring of 1954. By June, four of these six plants showed leaf symptoms typical of Pierce's disease (3) and by the first week of July, one additional plant

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showed symptoms. The sixth plant and all 10 control plants remained healthy during the 1955 season and up to July 1956, when they were destroyed.

Graft transmission -- In July 1955, two of the five plants from Meridian which showed symptoms of Pierce's disease were approach-grafted, each to a healthy Emperor plant. In December the approaching canes were severed, leaving a portion of the cane of diseased vine attached to the test plant. By mid-June in 1956 these two test plants showed typical symptoms of Pierce's disease, thus proving graft-transmissibility of the virus. Three of the five Emperor grape plants from Meridian were dead by November 1955 and the other two failed to leaf out in the spring of 1956.

Insect vector recovery -- Virus recovery tests with nymphs of sharpshooter leafhopper Draeculacephala minerva were run on three of the Emperor plants from Meridian which showed Pierce's disease symptoms. Non-viruliferous nymphs were reared from eggs on barley seedlings in a manner similar to that described by Freitag (2). Nymphs in the third instar were first caged on healthy Emperor grape or healthy alfalfa plants for control. The nymphs were then moved to one of the diseased Emperor plants from Meridian where they were caged for 3 days. Surviving nymphs were then caged for 3 to 4 days in sequence on a healthy grape plant and then on an alfalfa plant, or in the reverse order, in a manner similar to that described by Hewitt, Houston, Frazier and Freitag (4).

After 9 months' incubation by July 1956 all control plants had remained healthy. The Pierce's diseased Emperor plants from Meridian were all dead. In these tests Pierce's disease virus was recovered from two of the three diseased Emperor plants from Meridian and transmitted by vectors to two healthy Emperor grape rootings and to two healthy alfalfa plants as indicated in Table 1.

Table 1. Results of Pierce's disease virus recovery tests with nymphs of Draeculacephala minerva from three Emperor grapevines grown in the open at Meridian, Mississippi during the 1953 season and returned to Davis.

Test series	Sequence of plants upon which nymphs were caged	Number of nymphs	Number of days on plant	Condition of plant after 9 months incubation
1	Carignane	25	3	healthy
	Emperor #2[a]	18	3	dead
	Carignane	5	3	healthy
	Alfalfa	2	4	healthy
2	Emperor	25	3	healthy
	Emperor #3[a]	13	3	dead
	Emperor	9	3	Pierce's disease
	Alfalfa	4	3	dwarf
3	Alfalfa	25	3	healthy
	Emperor #4[a]	22	3	dead
	Alfalfa	8	3	dwarf
	Emperor	7	3	Pierce's disease
	Alfalfa	3	4	healthy
4	Carignane	25	3	healthy
	Emperor #3[a]	11	3	dead
	Emperor	5	3	healthy
	Alfalfa	3	4	healthy

[a]The Emperor vine numbers 2, 3 and 4 were from Meridian and showed symptoms of Pierce's disease.

Observations of grapes and alfalfa in Mississippi and Georgia -- During September 19 to 24, 1957, Wm. B. Hewitt visited the United State Horticultural Field Stations at Meridian, Mississippi and Fort Valley, Georgia. This trip was made at the invitation of Dr. John R. Magness, Acting Head, Fruit and Nut Crops Research Branch, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, for the express purpose of observing grapevines and alfalfa in experiments at these stations and for an exchange of experiences and ideas with the respective staffs.

Pierce's disease in grapes and its concomitant disease, the dwarf disease in alfalfa, were observed in seedlings and varieties of both muscadine and bunch grapes on the station grounds at Meridian, and in muscadine and bunch grapes and in alfalfa plants on the station grounds at Fort Valley. Enroute from Meridian to Fort Valley the disease was observed in experimental test plantings of both bunch and muscadine grapevines at Auburn, Alabama.

DISCUSSION

Though these tests involve relatively small numbers of plants they are positive evidence of the presence of Pierce's disease virus in the vicinity of Meridian, Mississippi. Possibilities are very remote that the vines became infected with the virus at Davis during the course of the experiment because there were no other cases of Pierce's disease observed among the control plants of these lots held at Davis or in other experimental grape and alfalfa plants in the glass house at Davis where these plants were kept. Also, no cases of natural spread of Pierce's disease were observed in the vineyards at Davis during the seasons of these experiments.

No explanation of the failure to recover Pierce's disease from Mississippi the first season of the tests, that is 1952, is attempted. Possibly the experimental numbers were too small, as suggested by the high mortality of test vines. This was especially so at State College.

Experiments demonstrating the presence of the virus in Leesburg, Florida (1, 7) and those of this paper showing the presence of the virus at Meridian, Mississippi, are strong support that the symptoms typical of Pierce's disease seen on grapevines in Alabama and on grapevines and alfalfa at Meridian and Fort Valley in September 1957 were caused by the Pierce's disease virus. Thus it would appear that since the virus is present in these widely separated places it could occur generally over much of the Gulf Coast area of the Southeastern United States.

CONCLUSIONS

Tests reported in this paper show that the Pierce's disease virus occurs in the vicinity of Meridian, Mississippi and that the virus transmitted to grape and alfalfa and produced symptoms typical of Pierce's disease and dwarf as they are known in California. The disease was also observed in Alabama and Georgia.

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THE PROBABLE HOME OF PIERCE'S DISEASE VIRUS

Wm. B. Hewitt[1]

Historical records indicate that Pierce's disease virus is not indigenous to California, but that the virus was probably introduced into the State. The widespread distribution of the virus in the United States Gulf Coastal Plain area, coupled with the fact that several Vitis species highly tolerant to the virus are indigenous to the same area, indicate that this is the probable home of the virus. Repeated failures of bunch grape plantings in the southern United States over many years have probably been the result of Pierce's disease virus. It also seems likely that the virus may have been introduced into California in grape wood from this area.

For many years Pierce's disease virus has been known to be widely distributed in California (6, 13). The virus has been identified in the variety Thompson Seedless (Vitis vinifera) near Carizzo Springs, Texas (13), in Florida (4, 23, 24), at Meridian in Mississippi and at Fort Valley in Georgia (15). The disease has also been observed in seedling bunch grapes and muscadine grapes at Auburn, Alabama (15). These localities spot the disease over much of the Coastal Plain areas of the United States and in many parts of California.

Pierce's disease was first identified in southern California near Pomona and Anaheim about 1884 (6, 20), in northern California in Napa County in 1887, in Livermore in 1888 (6), in the Sacramento Valley and in the Santa Clara Valley about 1900 (21, 22). Apparently the first report of the disease in the San Joaquin Valley was about 1921 (13). During the enphytotic of 1935-1941 the disease was found in most of the principal vineyard districts of California (13).

In California, historical records show that the Mission grape was first planted at the time of the building of the Spanish missions at San Diego in 1769, at San Gabriel in 1771, and subsequently at other missions as they were established in more northerly areas. These were without question the first plantings of the European grape Vitis vinifera L. in California (20). Plantings at San Gabriel grew well, they were extensively planted and apparently were the center of distribution of the variety to other missions. During the next 100 years there were no records of widespread destruction of grapes by disease in California.

Newton B. Pierce, in his bulletin "The California Vine Disease" (20), gives a thorough and very well-documented review of the introduction of the European vine into California, and the spread, growth, and development of the grape industry prior to 1884. The principal object of this review by Pierce was to establish that the European grape was extensively planted in California before 1884, that there were many very old plantings, and specifically, that there was no record of any previous destruction of grapevines by disease such as that which occurred between 1884 and 1900.

The only statement that I have found in historical records which might appear to conflict with the evidence that Pierce's disease did not occur in California for many years prior to 1884 is in a report by Ethelbert Dowlen published in 1890 (6). He was employed by the California State Viticultural Commission to study the "Anaheim vine disease". In this report Dowlen states, "The Anaheim disease has probably existed in the State for many years past. It was noticed in Napa County in 1887 and again in Napa and Livermore in 1889....". This reference to many years past appears to be an opinion, for no facts were given to substantiate it. It probably refers to a period of not more than 10 or so years because, in the summer of 1939, I talked with Mr. W. Shehon, the owner of a vineyard that had belonged to his father on Spring Mountain in Napa County, California, in which the disease was first identified in northern California. While in his vineyard Mr. Shehon identified the disease in some of the vines and stated that it was the "California Vine Disease" and that it had been identified on his father's place in 1887 by a man from the United States Department of Agriculture and several other people. Records show that L. F. Scribner of the United States Department of Agriculture and Pierre Viala, Professor of Viticulture at National School of Agriculture, Montpellier, France, along with others, visited the Napa Valley in 1887 and identified the disease in two localities (6, 27). Mr. Shehon further showed me the location in the vineyard where the disease first became apparent. It was in a block of vines adjacent to the Shehon mother nursery of "American" Vitis species that were introduced for the purpose of testing their resistance to Phylloxera. Mr. Shehon recalled that his father had introduced "wild grape" from nurseries in Missouri and Texas and possibly from other places. Mr. Shehon could not remember the date of the first evidence of the disease for, as he said, he was only a boy at the time. How-

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ever, he agreed that it was possible that the disease could have been in the vineyard for a few years without being recognized because of Phylloxera damage to the vines.

Although this experience was from Mr. Shehon's memory and is now reinterpreted from my notes of the conversation, the records (6, 27) indicate that the information was essentially correct. If so, then it is probable that the virus may have been introduced into this Napa County area along with "American" Vitis species from which it spread to native plants and to the European grapes growing nearby.

In California, Pierce's disease virus has been recovered from a wide variety of host plants, over 80 species in about 70 genera and 27 families of dicotyledonous and monocotyledonous plants (12, 13). The virus may have been introduced into California in any one or a number of different species of plants. It is also probable that the virus may have been introduced in wild grape from the Gulf Coastal Plain area of the United States, and that it may have been introduced a number of times in different localities. The virus spread because there were vectors present in California.

There are many records of introductions into California of named varieties of American grape species and of collections of cuttings of wild grapes from parts of Texas, Missouri, Arkansas, Florida, Nebraska and other States for testing their resistance to Phylloxera and their suitability as a rootstock for the European grape. Some of the largest introductions into northern California were made around 1876 and later (1, 5, 7, 16, 17, 19). However, none of these introductions can be specifically associated with the first outbreaks of Pierce's disease.

There were also many introductions of American grape varieties including the muscadines (Vitis rotundifolia Michaux) into southern California (3, 18, 20), but no records could be found of introductions that were directly associated with the first appearance of the disease near Anaheim and Pomona.

In the Southeastern Coastal and the Gulf Plain areas of the United States it is apparently well established that most bunch grapes do not survive for very many years after planting. Stoner (24) briefly reviewed the failure of bunch grape culture in Florida and showed degeneration of vines in the State to be caused by the Pierce's disease virus. Stoner, Stover and Parris (23) listed groupings of grape varieties that had been tested using longevity as an index of tolerance to degeneration. Stoner (24) and recently Crall and Stover (4) showed that the vectors of the virus in Florida belong to the same genera as some of those reported in California (10, 11, 14).

Hewitt, Loomis, Overcash and Parris (15) have shown that the virus is present in Meridian, Mississippi. In addition there are many early reports in the literature which record the failures of bunch grapes in many parts of the Gulf Coastal Plain area. These records attribute the failure to various causes. No attempt is made in this report to search out all such records. Only a few are mentioned to indicate the nature of the records of the failure in establishing satisfactory grape culture in the Gulf States. A report by Earl (8) in 1896 on grape culture in Mississippi states that "a careful selection of varieties is essential as all are not equally successful". He further wrote that none of the European or Vitis vinifera type grapes can be recommended. "They may grow well for a time, especially if trained against a building, but they are much troubled with mildew and other diseases....".

The following report from Alabama is a typical case history of bunch grape failure in many parts of the South Coastal Plain area. Bondurant and Clayton (2) report that six vines each of most of the 48 varieties of bunch grapes planted in 1886 were dead by 1893. In fact, the only survivors in the vineyard were a few vines of the varieties Concord, Delaware, Ives, and Perkins, of which 100 vines each had been planted originally. Another vineyard planted in 1889 had a few surviving plants in only 17 out of 78 varieties that were originally planted. In contrast, all vines in eight varieties of Vitis rotundifolia planted at the same time were growing well. The death of the bunch grape plants was attributed to bunch rot.

Earl and Austin (9) in 1900 report that this root rot was one of the most serious diseases of grapes. They showed that over 75 percent of 651 vines planted in 1894 were dead by 1900, but the root rot could not be attributed to any specific organism. There were only two varieties, Herbemont (Vitis bourquiniana Munson) and Rulander, out of 16 varieties planted that were entirely resistant.

Root rot is one of the symptoms characteristic of Pierce's disease of grapevines in California. The tops and roots die back at about the same rate. The last part of the vine to die is usually near the crown. The fact that the root rot in Alabama could not be attributed to a fungus pathogen is a factor favoring the hypothesis that Pierce's disease was responsible for

the death of the vines.

Stubbs, Burnette and Watson (25) in 1898, reporting on grape culture, stated that "over 100 varieties of grapes have been grown on the different stations. Gradually, one by one, the number is reduced and today but few are found suited to different localities of the state".

Munson in his book on "Foundations of American Grape Culture" published in 1909 (19) recognized that few northern varieties grew successfully in the Gulf regions.

Vitis species reported to have resistance to Pierce's disease are V. rupestris (Schede) Linn. (21), V. champini Planchon (19), V. coriacea Shuttleworth (19, 23, 24), V. simpsoni Munson (19), and V. rotundifolia Michaux (13). There are probably other species such as V. riparia that have resistance to Pierce's disease but have not been tested.

The resistance of V. rupestris to Pierce's disease was noted by Pierce (21) when the disease was prevalent in the Santa Clara Valley of California. He states that some susceptible varieties such as Mataro (V. vinifera) are quite resistant when grafted onto the St. George (V. rupestris). I have also observed that selections of St. George appear to be resistant and in some cases apparently immune to the virus. The variety Grenache (V. vinifera) has apparent klendusity to the disease and when grafted onto St. George rootstock the resistance appears to increase. In contrast, the variety Green Hungarian (V. vinifera) and many others are very susceptible when grown on their own roots and also when grown on St. George rootstocks.

Munson (19) pointed out the resistance of V. champini to Pierce's disease. He states on page 39 of his book, ".... and it is one of the most resistant species against the Phylloxera and Anaheim disease". During my recent visit at Meridian (15), N. H. Loomis pointed out that V. champini grows very well without apparent symptoms of Pierce's disease, and when used as a rootstock, it also lengthens the life of the variety grafted upon it.

The Florida species V. coriacea, V. simpsoni, and perhaps others, have been shown to be resistant to Pierce's disease in Florida (23, 24). Munson (19) wrote of V. coriacea, "It appears to me that this species offers good material upon which to base a valuable strain of table and wine grapes for the Gulf regions where few northern varieties succeed".

Some varieties of V. rotundifolia showed good resistance to Pierce's disease in California plantings. From observations made on a recent trip into the Southern States (15), it appears that seedlings of crosses between varieties of V. rotundifolia show varying degrees of resistance to the disease. Some plants are susceptible, nevertheless many are symptomless carriers of the virus and others may have been immune. I recovered the virus, however, from a resistant plant listed as V. rotundifolia under test in a planting on the campus of the University of California at Los Angeles in 1941 that did not have any symptoms of Pierce's disease.

The varieties Lenoir (Jaquez) and Herbemont and their seedlings which Munson (19) considers varieties of V. bourquiniana Munson will grow very well in areas of the South where many of the other varieties of bunch grapes degenerate as pointed out by Stubbs, 1898 (25), Munson (19), and by Earl and Austin (9). Pierce (22) found Lenoir resistant to Pierce's disease in California.

These different species of grapes which have resistance to Pierce's disease virus are native to regions within the vast Coastal Plain area (19, 27, 28). Many other Vitis species native to other regions of the United States and Europe such as labrusca, californica, arizonica, and vinifera, are not resistant to Pierce's disease. This report makes no effort to include all grape species, indeed, there are many species that are yet unclassified with respect to their susceptibility to Pierce's disease.

CONCLUSION

The long history of over a hundred years' culture of European grape varieties in southern California prior to the first appearance of Pierce's disease at Anaheim and Pomona in 1884 is fairly good evidence that the virus which causes this disease was not native to southern California. There is a possibility that the virus might be native to the northern coast counties of California, but since Vitis californica, native to this area, is susceptible to Pierce's disease and the vines degenerate and die out with the disease, it is not likely the virus had been there for a long period.

If we accept the premise suggested by the writings of Vavilov (26) that Vitis species resistant to the disease most likely developed in areas native to the virus, then, since the Vitis species native to the United States Gulf Coastal Plain area are the only ones known to have resistance to Pierce's disease and since Pierce's disease virus is known to be present in these

areas, it is probable that Pierce's disease virus is native to the Gulf Coastal Plain areas of the United States.

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A NOTE ON APHID TRANSMISSION OF A FEATHERY MOTTLE VIRUS OF SWEETPOTATO

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Two non-persistent viruses of the feathery mottle type were isolated, by aphids, from sweet potato plants at Berkeley, California. These could be distinguished in severity on Jersey variety sweet potato, and even more readily on the Brazilian Morning Glory, Ipomoea setosa Ker. Also, the two isolates, designated as severe feathery mottle and mild feathery mottle, were separable on the basis of pathogenicity, or lack of it, to 10 convolvulaceous species. A more detailed study of the severe isolate, with special reference to the vector-virus relationships, is currently being conducted by the junior author.

Webb and Larson (2) transmitted feathery mottle of sweet potato, with the green peach aphid, Myzus persicae (Sulzer), as the vector. The aphids were given an access feeding period of 3 days on the virus source plant and a test feeding period of 3 days. Groups of 20 viruliferous aphids were used to inoculate each of 10 plants of several varieties of sweet potatoes.

In the present investigation the severe isolate was efficiently transmitted by single apterous green peach aphids when Ipomoea setosa was used as a test plant. Evidence that it is a non-persistent virus is based on data that clearly indicates relationships similar to those reported by Sylvester (1) for other non-persistent viruses. Pre-acquisition fasting, coupled with a short acquisition feeding, increased the transmission efficiency of the green peach aphid. Serial transmission trials, using the green peach aphid as the vector, indicated that the virus charge was lost rapidly, and transmission decreased as post-acquisition fasting time was increased.

In addition to the green peach aphid, the celery aphid, Aphis apii Theob., and the melon aphid, Aphis gossypii Glover, proved to be vectors of the virus, although somewhat less efficient.

Host-range studies using the green peach aphid as the vector indicated that only plants of the family Convolvulaceae were susceptible to the virus. Seven species of the genus Ipomoea and one species of the genus Quamoclit were experimentally infected.

Symptoms of severe feathery mottle developed on young Jersey variety sweet potato plants in 6 to 13 days with a mean of 8 days after aphid inoculation. The primary symptoms consisted of small bright yellow spots or streaks which occurred in a random fashion over the leaf. Soon the spots became faint, enlarged. Eventually they coalesced to form chlorotic areas bordering the main veins, spreading rapidly to produce typical vein feathering. Under high temperature conditions and possibly high light intensity, the foliar symptoms became masked about 2 weeks after first appearance. Definite symptoms could be distinguished only in the shaded portions of the lower interior leaves of large infected plants. In addition, the infected plants were somewhat stunted.

On Ipomoea setosa the symptoms appeared in 5 to 8 days, with a mean of 6.5 days after aphid inoculation. When the plants were inoculated in the cotyledon stage, the primary leaf exhibited a clear mosaic-type pattern and the leaf tended to curl upward slightly at its base. The second leaf, also mottled, was slightly darker in color and blister-like over the entire surface. The third and subsequent leaves were paler in color, more severely dwarfed, and strongly twisted, with the mid-rib and the main veins cleared and chlorotically banded. The infected plants were severely stunted and possessed a much reduced root system.

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TOMATO RINGSPOT VIRUS ASSOCIATED WITH STUNT OR STUB HEAD DISEASE OF GLADIOLUS IN FLORIDA[1]

R. F. Bozarth[2] and M. K. Corbett[3]

Summary

Tomato ringspot virus (Annulus zonatus Holmes) was isolated from gladiolus plants that had short flower spikes ("stub head") and were slightly stunted. The virus isolates were identified by means of differential hosts and by immunological studies with strains of tomato and tobacco ringspot viruses. This is believed to be the first report of natural infection with tomato ringspot virus in Florida.

The disease stunt or stub head has been prevalent in gladiolus growing areas for many years. Diseased plants are distinguished by flower spikes that are much shorter than normal and by a slight stunting of the entire plant. Mottling, streaking, and distortion of the foliage do not necessarily occur on stunted plants. This disease, of apparent wide distribution, may be responsible for much of the decline in vigor of older varieties; thus in importance it may surpass the other known diseases of gladiolus.

In an investigation to determine the viruses present in Florida-grown gladiolus, stunted plants were indexed. This paper is concerned with the identification of the viruses isolated from stunted plants.

The first description of a possible virus disease of gladiolus was by Dosdall (7) in 1928. It was not until 1946 that White (19) identified cucumber mosaic virus as being the causal agent of a disease of gladiolus. McWhorter et al. (11) reported bean yellow mosaic virus to be wide spread in gladiolus in the United States. Since 1947, other viruses have been isolated from gladiolus. Among these are the viruses of common bean mosaic (18), tobacco ringspot (1, 3, 4), tomato ringspot (18), aster yellows (15), western aster yellows (10, 16), and additional strains of bean yellow mosaic and cucumber mosaic (2, 3, 6, 8, 12).

MATERIALS AND METHODS

Diseased Material: Virus isolates were obtained from gladiolus plants that had short spindly flower spikes. These short flower spikes, which are often referred to as stub heads (Fig. 1), had four to five florets per spike. The leaves of affected plants were smaller in size than the normal. Plants of the variety Phantom Beauty, in addition to being stunted, exhibited many small chlorotic lesions that tended to elongate into transverse chlorotic flecks terminated by the leaf veins.

Virus Isolates and Methods of Inoculation: Virus isolates 45 and 50 were obtained from stunted plants of the gladiolus variety Elizabeth the Queen. Isolate 35 was obtained from stunted plants of the variety Bridal Orchid. These plants were collected from the gladiolus-growing areas of East Palatka and Bradenton, Florida. Tobacco ringspot virus (Annulus tabaci Holmes) (TobR), American Type Culture Collection Number 16, and tomato ringspot virus (Annulus zonatus Holmes) (TomR), ATCC No. 13, obtained from A. F. Ross, were used for comparative inoculations and immunological studies. All viruses were maintained in plants of Gomphrena globosa L. or of Nicotiana tabacum L. variety Turkish. Leaves and flower parts of gladiolus, when available, were triturated with a mortar and pestle, and the

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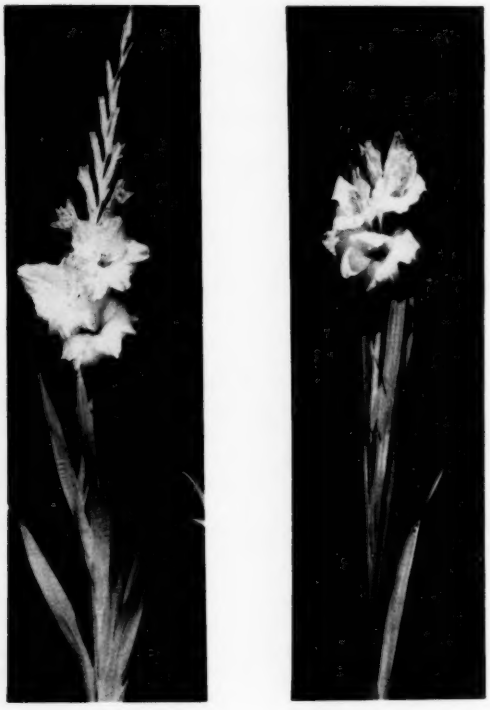

FIGURE 1. Left -- healthy gladiolus plant of the variety Elizabeth the Queen. Right -- plant of the same variety showing short flower spike.

juice thus obtained was used for inoculum. All mechanical inoculations were made by the gauze-pad method. Leaves were dusted with 500-mesh carborundum, then inoculated and immediately rinsed with tap water.

The green peach aphid, Myzus persicae (Sulz.), was obtained from field-grown plants of Lupinus luteus L. and transferred by means of a camel's-hair brush to plants of Solanum tuberosum L. Colonies thus obtained were used for all insect transmission trials. All experiments were conducted in screened greenhouses that were periodically sprayed with parathion.

HOST RANGE

Various hosts were mechanically inoculated separately with the three virus isolates from gladiolus and with the known strains of TobR and TomR. Plants of Turkish and White Burley tobacco developed local lesions approximately 4 days after inoculation; these were followed by severe necrotic oak leaf patterns on noninoculated leaves and eventual recovery (Table 1). Isolate 45 did not move systemically in these hosts. Inoculated plants of N. glutinosa L. developed local lesions, but the virus was not recovered from the noninoculated leaves.

Plants of Datura stramonium L. inoculated with TobR showed necrotic local lesions approximately 4 days after inoculation, followed by systemic spread and the appearance of oak leaf patterns on noninoculated leaves. Plants of D. stramonium inoculated with TomR or with isolate 50 developed a chlorotic mottle rather than oak leaf patterns.

Plants of Gomphrena globosa developed necrotic local lesions 3 days after inoculation with isolates 45, 50, or TomR. Isolate 35 and TobR caused no symptoms on the inoculated leaves of G. globosa. All isolates moved systemically and induced a mottle in this host.

All isolates induced local lesions and necrosis of noninoculated leaves on inoculated plants of Gypsophila elegans Bieb.

Plants of Antirrhinum majus L. developed necrotic local lesions when inoculated with any of the viruses except TomR. Isolates 45 and 50 caused a mottle on noninoculated leaves, and TobR caused tip necrosis following systemic movement.

Table 1. Symptoms induced by three viruses isolated from gladiolus, by tomato ringspot virus (TomR), and by tobacco ringspot virus (TobR), in certain differential hosts.

	Symptoms induced by indicated strain [a]				
	Gladiolus isolates				
Host	35	45	50	TomR	TobR
Nicotiana tabacum					
var. Turkish	R/S	R	R/S	R/S	R/S
N. tabacum					
var. White Burley	R/S	R	R/S	R/S	R/S
N. glutinosa	R	R	R	(R)[b]	(R)[b]
Datura stramonium	L	L	L/M	L/M	L/S
Vigna sinensis					
var. Blackeye	L/T	L/T	L/T	L/T	L/T
Cucumis sativus					
var. Marketer	L/M	L/M	L/M	L/M	L/M
Gomphrena globosa	M	L/M	L/M	L/M	M
Zinnia elegans var.					
Lilliput Golden Gem	M	M	M	M	M
Gypsophila elegans	R/SN	R/SN	R/SN	R/SN	R/SN
Antirrhinum majus	R	R/M	R/M	O	R/T

[a] S = oak leaf patterns on noninoculated leaves, L = local lesions, T = tip necrosis, R = ringspot, M = mottle, SN = necrosis on noninoculated leaves, O = no symptoms.
[b] Reported by Price (14).

Plants of Cucumis sativus L. developed chlorotic local lesions and a systemic mottle when inoculated with any of the viruses. A light systemic mottle was produced by all viruses in inoculated plants of Zinnia elegans Jacq. In inoculated Blackeye cowpea plants (Vigna sinensis (Torner) Savi), all viruses induced necrotic local lesions followed by systemic movement and tip necrosis.

IMMUNOLOGICAL RELATIONSHIPS

Cross-protection tests were conducted with TobR, TomR, and the gladiolus ringspot isolates. Two separate experiments were conducted.

1) Tobacco Ringspot as the Protecting Virus: Fifteen young Turkish tobacco plants were inoculated with TobR and 15 plants were maintained as controls. When the inoculated plants had recovered from the acute stage of the disease, tip cuttings of each plant were dipped in Rootone and rooted in vermiculite. After root formation, they were transplanted to 4-inch clay pots containing composted soil. When all plants were established, each plant grown from an infected cutting was tested for TobR; all were found to be infected. Four healthy and four diseased plants were inoculated with a challenge virus; the viruses used were isolates 35, 45, and 50, TobR, and TomR.

No local lesions were produced on plants reinoculated with TobR. The control plants and plants systemically infected with TobR all developed numerous local lesions following inoculation with the gladiolus isolates or with TomR. This indicates a lack of immunological relationship between the gladiolus viruses and TobR.

2) Tomato Ringspot as the Protecting Virus: Sixteen Turkish tobacco plants were inoculated with TomR and 16 plants were retained as controls. When the inoculated plants had recovered from the severe stage of the disease, four healthy and four diseased plants were each inoculated with a challenge virus (gladiolus isolates 35, 45, or 50, or TobR).

The control plants inoculated with isolate 35, 45, or 50 or with TobR developed many necrotic lesions on the inoculated leaves. The plants systemically infected with TomR when inoculated with isolate 35, 45, or 50 did not develop lesions on the inoculated leaves. These positive tests indicate relationships between the gladiolus isolates and TomR. The plants systemically infected with TomR when reinoculated with TobR developed lesions on the inoculated leaves.

TRANSMISSION

Attempted Transmission by Means of Aphids: Smith and Brierley (17) reported aphid transmission of tobacco ringspot virus from gladiolus to gladiolus. Although the insect vector of tomato ringspot virus in unknown, attempts were made to transmit the gladiolus isolates 35, 45, and 50 by means of the aphid Myzus persicae. Systemically infected plants of Gomphrena globosa were used as the source plants, and gladiolus clone A57-3, grown from half corms (2), as the test plants. Plants produced from the opposite half of each test corm were used as the controls. Single aphids were given timed acquisition feeding periods of 12 to 20 seconds and transferred to gladiolus test plants, five aphids per plant. Sixteen test plants were used for each test. No symptoms developed on the test plants after 8 weeks, and the virus could not be recovered by mechanical inoculation of Turkish tobacco plants.

Attempted Mechanical Inoculation of Gladiolus: Attempts were made to reproduce symptoms by inoculating 25 gladiolus test plants of the clone A57-3 with isolate 50. Ten weeks after inoculation the test plants were symptomless and the virus could not be recovered by inoculation to Turkish tobacco plants.

DISCUSSION

The identification, on the basis of host range and cross-protection tests, of the virus associated with stunting of gladiolus as a strain of tomato ringspot virus constitutes the first report of this virus from a crop grown in Florida.

Tomato ringspot virus was first described by Price (13). Although its experimental host range is rather extensive, it has been found to infect only a few species of plants in nature. Hildebrand (9) reported a ringspot disease of currants caused by tomato ringspot virus,

Brierley (5) reported this virus as causing a ringspot disease of florists' hydrangea, and Snow (18) reported its occurrence in diseased gladiolus grown in Oregon.

The effect of tomato ringspot virus on the gladiolus crop remains uncertain. Snow (18). isolated the virus from plants with chlorotic bands and dark green rings. The plants from which tomato ringspot virus was isolated during this investigation were slightly stunted and had symptoms commonly referred to as stunt or stub head. Attempts to transmit tomato ringspot virus from various source plants to gladiolus by mechanical means and aphids were not successful. No visible symptoms of infection occurred on the inoculated gladiolus and the virus could not be recovered.

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A LEAF CURL DISEASE OF TOBACCO OBSERVED IN MARYLAND[1]

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Summary

In 1950, a leaf curl of tobacco with symptoms similar to the common leaf curl was observed in Maryland. The disease was graft transmitted to other tobacco varieties, to tomato and Datura stramonium. In attempts to transmit the disease by serial grafts, it became progressively less severe. No symptoms appeared after three serial graft transmissions in tobacco and tomato. By 1951, all symptoms of the disease had disappeared.

INTRODUCTION

While surveying tobacco fields late in the summer of 1950 a number of dwarfed tobacco plants with extreme curling of the leaves were found in two fields. In one field of 3/4 acre, from which most of the tobacco had been harvested, suckers with curled leaves were found growing from the stubble. Some of the uncut tobacco also had the leaf curl. The infection apparently started from a common center and spread outward. About 20 percent of the plants were infected. The second, larger field of about 10 acres was approximately 200 yards from the small field. Approximately 150 plants along the edge nearest the small field had leaf curl. Surveys in the area through the years 1951 to 1955 did not bring to light any new infections. Neither tomatoes in the growers' garden nor datura plants near the fields showed signs of the disease.

The disease (Morgan and McKinney, 3) bore a strong resemblance to leaf curl as described by Storey (5), Pal (4), Vasudeva (9), and Wolf (10). Valleau (8) reported a leaf curl from Kentucky. Since a leaf curl described by other investigators was transmitted by species of white flies of the genera Bemisia, Trialeurodes, and Aleurotrachelus, a thorough search of the area was made for the fly. None were found. Sharp and Wolf (7) were able to study particulate material found in the sap from tobacco with leaf curl. They state that the particles were spherical and of one kind and probably were the virus of leaf curl.

SYMPTOMATOLOGY

Field symptoms consisted of dwarfing and extreme curling of the leaves with marked reduction in leaf size (Fig. 1A and 1C). Most of the plants observed were suckers from tobacco stubble. Many of the dwarfed suckers had all the leaves curled and presented a rosette appearance (Fig. 1A). The mature, small, uncut tobacco had symptoms of the disease in the tops and on suckers, indicating that the disease had appeared late in the season. The dwarf condition was due to shortening of internodes and resulted in a short, stubby plant with many leaves, up to 30 in some, and a thickened stalk (Fig. 1C).

The first symptoms of the disease appeared in the new leaves which were extremely curled and wrinkled. The interveinal areas were crinkled, rugose, thick, and brittle. Early color of the leaf was a light green turning to dark green later. The tips of the leaves and the margins curl downward and inward. Figures 1A and 1F show an extremely curled leaf pattern. In larger, older leaves the veins were knotty, bumpy, and dark green in color. This knotty appearance most nearly approached the enations described by Storey (6). Many leaves were twisted around several times and hooked at the tips, giving a corkscrew appearance (Figs. 1A, 1C, 1E, and 1F). In one instance, several leaves of a plant had bent downward and twisted completely around the stalk two to three times. Leaves, apparently when they were older, showed downward crimping of the margins and tips (Fig. 1C). In no case was apical dominance diminished so as to allow branching as described by Wolf et al. (10). No vein banding or clearing of veins or mottling of leaves occurred as described by Vasudeva (9), Wolf et al. (10), and Pal (4). No large cup-like outgrowths appeared as described by Matsumoto (1). Since some

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FIGURE 1.

A. Shows leaf curl symptoms in most leaves of the tobacco plant.
B. Leaf curl in tomato. Top healthy; bottom plant diseased.
C. Shows stunting in tobacco.
D. Dieback of tobacco leaves.
E. Shows new leaves of grafted plant with leaf curl. (see G.)
F. Datura stramonium with leaf curl.
G. Shows leaf curled tobacco grafted to healthy plant which later developed leaf curl (see E. above).

of the features characteristic of the disease as described by other workers were either absent or present in a reduced form, the leaf curl described here was probably a mild form of the disease. All plants brought to the greenhouse produced new terminal growth with leaves free from the leaf curl symptoms. Die-back of infected leaves was observed in the greenhouse but not in the field. Yellowing followed by necrosis at the tip of the leaf or at the end of a vein, gradually extending back toward the midrib and finally to the petiole, resulted in death of some of the infected leaves. Some of the stalks of the leaf-curled plants curved in a zigzag manner either along the entire length of the stalk or only a portion of it. By June of 1951, all the surviving plants brought into the greenhouse in the fall of 1950 were producing growth normal in appearance.

MATERIALS AND METHODS

Leaf curled plants were brought from the field and potted in the greenhouse. Tobacco varieties used in transmission studies were Maryland Catterton, Turkish Samsoun, and a mosaic-resistant variety, 448-A. Tomato plants of the Marglobe variety and Datura stramonium were also used in transmission tests. The only successful methods of transmission were by inarch and wedge grafts (Morgan, 2).

RESULTS

Five severely leaf curled plants from the field were used in the grafting series. Eight 448-A plants were grafted with diseased scions and later showed symptoms of leaf curl. Also, the 448-A plants showed a pronounced zigzaging of the stalks. Normal appearing leaves ultimately grew out above the curved portion of the stalk. Symptoms resulting from grafting the infected 448-A plants to healthy 448-A plants were less severe and after the third serial graft all signs of the disease disappeared.

The first grafts made to Catterton (Figs. 1E and 1G), Turkish Samsoun, and 448-A tobacco required about 40 days for the symptoms to appear. By cutting the tops and large leaves from the grafted plants, the time of appearance of the curled leaves was reduced to 13 to 22 days. As the new leaves appeared they became curled. Healthy plants of the same varieties grafted with healthy scions and with tops and leaves removed did not produce curled leaves.

The disease was graft-transmitted to seven Turkish Samsoun and four Maryland Catterton plants. After symptoms had reached a maximum of severity and curled leaves had started to die, the new leaves that formed usually were not curled. All tobacco plants to which the disease had been transmitted "grew away" from the disease or died.

Transmission of the leaf curl to tomato followed much the same pattern as in tobacco. No further symptoms appeared after the third serial graft from tomato to tomato. The leaf curl was transmitted from tobacco to three tomato plants (Fig. 1B). The leaves of both the grafted plant and the healthy plant (Fig. 1B) were removed at the time the grafts were made. Both plants were the same age. The difference in size of plants and leaves is due to stunting of the grafted plant by the leaf curl. Symptoms appeared 3 to 6 weeks after grafting on the new leaves of tomato.

Of the three datura plants grafted with the curled tobacco scions, one showed symptoms of the disease 2 months after the scion was attached to the plant (Fig. 1F). All leaves were severely curled and no new growth appeared. The datura plant died 4 months after the diseased tobacco was grafted to it.

Eight of the leaf curled plants from the field were allowed to grow to maturity. Six of these were so severely affected that they did not produce an inflorescence. Two flowered and produced seed. The flowering heads were in compact clusters due to short internodes and pedicels (Fig. 1C). Seed from these curled plants were germinated and produced normal plants. Isolations from the curled plants did not yield any organism associated with the diseased plants.

DISCUSSION

It was not possible to determine the exact cause of the disease, but in the absence of any causal organism or condition that may have incited the leaf curl in Maryland, it seems most probable that the leaf curl was caused by a virus. Although the disease resembled tobacco leaf curl as described in the literature in some respects, some characteristics indicate that

it may have been a mild form of the disease. No definite enations appeared at any time in the field or greenhouse. No vector was found and, if one ever had been present, it disappeared from the field before the disease was discovered. The disease was not maintained by grafting, suggesting the possibility that a vector may be necessary for maintenance of the causal virus. The disease was most severe in the first graft transmission to tobacco, tomato, and datura. In attempts to transmit the disease by serial grafts, it became progressively less severe. No symptoms appeared after three serial graft transmissions in tobacco and tomato.

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STABILITY OF WHEAT STREAK MOSAIC VIRUS IN DESICCATED TISSUE[1]

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The infectivity of many plant viruses can be maintained when stored in finely cut host leaf tissue desiccated over calcium chloride at temperatures of 1° to 2° C (3, 4). Forty samples of wheat streak mosaic virus were collected in Kansas and the Great Plains from 1950 until 1955. After desiccation and storage over calcium chloride at 1° C in half-pint glass jars, they were tested for infectivity each fall from 1954 through 1956. This paper describes the level of infectivity of these wheat streak mosaic virus samples during the test period.

MATERIALS AND METHODS

Usually, young susceptible wheat plants were inoculated with juice from field infected plants, using the common carborundum leaf wiping technic (1). After confirmed infection the leaves of these young diseased plants were then chopped into very thin pieces and desiccated. Occasionally, diseased plants were desiccated immediately when brought in from the field. A layer of calcium chloride was placed over the bottom of the glass jars. On top of this was a layer of cotton and then a filter paper cup was pushed down on top of the cotton. The chopped leaf tissue was placed in these cups and the jars were sealed with airtight lids and labeled.

Tissue samples from these jars were tested for infectivity of the virus by soaking a little desiccated leaf tissue in a small amount of water in a mortar and then grinding it thoroughly with a pestle. Young Pawnee wheat seedlings or Agrotricum plants (Agropyron elongatum x wheat, Kansas row No. 52-6605) were inoculated and observed for typical wheat streak mosaic virus symptoms as described by McKinney (2), Sill and Connin (6), and Schmidt et al. (5). In doubtful cases inoculations to Golden Giant sweetcorn were also made (7).

RESULTS

With one exception, all samples were infective in the 1954 and 1955 tests, the range of infectivity on wheat being 56 to 100 percent (Table 1). Seven samples were either completely inactivated or their infectivity was extremely lowered in the 1956 test (Table 1). These samples and some others which produced less than 60 percent infection in Pawnee wheat did not usually incite any local lesion production on the Agrotricum. By contrast, those samples which produced more than approximately 60 percent infection on Pawnee wheat in the 1956 test typically produced local lesions on the Agrotricum. Several samples desiccated in 1952 were nearly inactivated by 1956. Among the 1953 samples two were much reduced in infectivity, while others maintained 30 to 100 percent of the original infectivity.

DISCUSSION AND SUMMARY

Some wheat streak mosaic virus leaf samples were still infective after 5 years of desiccation and storage at 1° C. The sudden drop in the percentage of infection of some samples in the 1956 tests as compared with the 1955 tests was probably due to the accidental disconnection of the refrigerator during August, 1956, for about 10 days. The room in which the refrigerator was kept had a constant temperature of about 80° F and all samples were therefore exposed to that temperature for about 10 days. Even so, most of the samples were still infective in 1956, indicating that desiccated samples are able to withstand considerable abuse.

When isolates had lost 40 percent or slightly more of their infectivity on Pawnee wheat, no local lesions were produced on the Agrotricum (Agropyron x wheat hybrid, Kansas row No. 52-6605), indicating that the latter host is more difficult to infect and may be useful only in identification when highly infective virus sources are used.

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Table 1. Level of infectivity of 40 desiccated wheat streak mosaic virus samples on wheat and an Agrotricum local lesion hybrid.

Collection code name	Year of desiccation	Percentage of infection in Pawnee wheat		1956 reaction of Agrotricum, Kans. Row No. 52-6605
		1955	1956	
AK '52	1952	63	60	--
CK '52	1952	100	50	--
HK '52	1952	69	10	--
MS '52	1952	94	10	--
NG '52	1952	75	10	--
SC '52	1952	--	20	--
CK_2 '53	1953	69	50	--
Chks-1	1953	88	60	--
EK	1953	88	30	--
GCK	1953	94	50	--
HK-2	1953	100	30	--
MI(2)	1953	100	60	--
NPK	1953	88	50	--
NG_2	1953	80	40	--
Set V.S.	1953	100	100	LL
RL-1	1953	93	50	LL
S8-52-3	1953	56	10	--
S1-52-3	1953	67	40	--
SC '53	1953	100	10	--
UK '53	1953	100	70	LL
A_3 '54	1954	100	0	--
A_5 '54	1954	87	80	LL
GCH '54	1954	100	100	LL
Oats (New Cambria)	1954	100	100	LL
SD-1	1954	100	100	LL
SD-2	1954	100	100	LL
Wooderson'54	1954	100	50	--
WSM-1	1955	100	100	LL
WSM-2	1955	100	100	LL
WSM-3	1955	100	100	LL
WSM-4	1955	100	100	LL
WSM-5	1955	88	80	LL
WSM-6	1955	100	100	LL
WSM-7	1955	88	100	LL
WSM-8	1955	100	100	LL
WSM-9	1955	94	90	LL
WSM-10	1955	100	100	LL
WSM-11	1955	100	100	LL
WSM-12	1955	88	88	LL
WSM-13	1955	94	100	LL

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THE PERSISTENCE OF SYSTOX IN OAT PLANTS AND ITS EFFECT ON THE TRANSMISSION OF THE BARLEY YELLOW-DWARF VIRUS

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Summary

The systemic insecticide, Systox, used on oats as a soil drench, as a foliage spray, or as a seed treatment protected the treated plants against aphid infestation for varying lengths of time. It gave longer protection when applied as a soil drench than when applied as a foliage spray. Seed treatment gave intermediate protection. With the soil drench method, plants became toxic to aphids a few hours after the application of Systox. While it protected the plants from aphid infestation for some time, Systox did not give protection from barley yellow dwarf infection.

The systemic insecticide, Systox[2], is absorbed by the leaves, roots, and stems (1, 4) and transported to other parts of the plant. It has been used on a variety of host plants (1, 3, 4, 5, 6, 7, 9, 10) to control thrips, mites and aphids.

Roland (10) reported that Pestox III and Systox-sprayed fodder beets became infected by the beet yellows virus in spite of the aphids being rapidly killed after the introduction of the systemic insecticides. McLean (8) also found that some aphids were able to transmit the yellows virus to insecticide-treated beets before the aphids were inactivated. He found that demeton-sprayed leaves were lethal to aphids for 18 to 24 days after application.

The purpose of this study was to determine the persistence of Systox in oat plants when applied by different methods and its effect on the transmission of the barley yellow-dwarf virus in greenhouse tests.

MATERIALS AND METHODS

All plants were grown in greenhouse compost in 4-inch pots. The variety Clintland was used. Each experiment was carried out at least two times.

Aliquots of four different concentrations of the liquid systemic (Systox in 21.2% liquid form) were applied either as a soil drench or as a foliage spray on young oat plants. The concentrations used were 1:200, 1:400, 1:800, and 1:1600.

When applied as a soil drench, 50 millileters of the solution were poured on each pot with five young oat plants. Immediately after application and at weekly intervals thereafter, aphids were transferred to the treated plants.

When plants were sprayed, all the above-ground parts were wet to the point of run-off. A good spray coverage was insured by rotating the pots. Aphids were introduced as soon as the sprayed material had dried, and daily thereafter.

When applied as a seed treatment, IsoSystox as a 50% wettable powder on carbon was used at the rate of 0.5, 1, 2, and 4 grams per 100 grams of oat seeds. The dust and seeds were shaken in a 100-ml flask until all the dust that could adhere to the seeds was evenly distributed. The dusted seeds were then planted in soil. Daily tests to determine the toxicity of Systox in the plants arising from the treated seeds were made after they had attained a height of 1 inch.

Apple-grain aphids (Rhopalosiphum fitchii Sand.), one of the vectors of the yellow-dwarf virus, were used for bioassays and as vector for the virus. Tests for persistency terminated as soon as the aphids were found to be alive 48 hours after being placed on treated plants. Plants were not removed from the soil when they were tested for toxicity to the aphids.

The effect of Systox on the transmission of the yellow dwarf virus was also studied. Con-

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[2] Demeton.

Table 1. Persistence of Systox in oat plants when applied as a soil drench as determined by bioassay using apple grain aphids (Rhopalosiphum fitchii Sand.)

Concentration	Survival of aphids at given intervals (days) after treatment							
	7	14	21	28	35	42	49	56
1:200	0	0	0	0	0	0	+	+
1:400	0	0	0	0	0	+	+	+
1:800	0	0	0	0	0	+	+	+
1:1600	0	+	+	+	-	-	-	-
Control	+	+	+	+	+	+	+	+

0 = all aphids dead. + = some aphids alive. - = no tests made.

Table 2. Persistence of Systox in oat plants when applied as a foliage spray.

Concentration	Survival of aphids at given intervals (days) after treatment							
	1	2	3	4	5	6	7	8
1:200	0	0	0	0	0	0	0	+
1:400	0	0	0	0	0	+	+	-
1:800	0	0	+	+	-	-	-	-
1:1600	0	+	+	-	-	-	-	-
Control	+	+	+	+	+	+	+	+

centrations of 1:200, 1:500 and 1:1500 were used as soil drenches. Young oat plants treated with the systemic were colonized with viruliferous aphids at various intervals. Five aphids were allowed to feed for 1 day on each of the treated plants and the controls. Aphids were removed with a malathion spray.

RESULTS

The range of toxicity where Systox was applied as a soil-drench is shown in Table 1. Toxicity to aphids in plants treated with a 1:200 concentration lasted up to 42 days from the time of application. At this concentration the solution proved phytotoxic, causing burning of leaves, stunting of growth and eventual death of plants. Complete protection against aphid infestation of treated plants lasted up to 35 days from the time of treatment at concentrations of 1:400 and 1:800. At 1:1600 concentration, persistency in the treated plants was only 7 days.

When plants infested with aphids were treated by the soil drench method at a concentration of 1:200, the aphids were observed to die 2 hours later. When aphids were placed on the treated plants just after application of the 1:200 concentration, they also died 2 hours later.

Table 3. Persistence of toxicity in oat plants from Iso Systox treated seeds.

Grams dust per 100 g. seed	Survival of aphids at given intervals (days) after seeding							
	12	15	17	19	21	23	25	27
4	0	0	0	0	0	0	0	+
2	0	0	0	+	+	+	+	+
1	0	0	0	+	+	+	+	+
0.5	0	+	+	+	+	-	-	-
Control	+	+	+	+	+	+	+	+

Table 4. Transmission of yellow-dwarf virus to Systox treated oat plants (soil drench).

Concentration	Per cent diseased plants when inoculated at intervals (days) after treatment				
	1	2	3	5	6
1:200	62	90	90	70	100
1:500	80	--	91	100	86
1:1500	63	45	96	87	80
Control	92	--	100	86	100

The vapor effect of the insecticide, which may have contributed to the death of the feeding aphids in the preceding tests, was superficially worked out. Aphids on an untreated plant exposed over a 1:200 concentration contained in a petri dish 4 inches below were observed to die 2 days later. It appeared that the aphids were affected more quickly by the systemic action than by the vapor. It was reported by David and Gardiner (3) that aphids on a bean leaf were killed in 1 day when placed 1 1/2 inches over a 1% Systox solution.

The range of Systox toxicity when applied as a foliage spray at different concentrations is shown in Table 2. At 1:200, complete protection was obtained for 7 days, at 1:400 for 5 days, at 1:800 for 2 days.

By treating the seeds with the IsoSystox, the establishment of aphids on plants from these seeds was prevented for a considerable length of time (Table 3). Complete control of aphids lasted up to 12 days from the time of seeding when seeds were treated at the rate of 0.5 grams. Both the 1- and 2-gram rates gave protection for 17 days from the time of seeding. The 4-gram rate of application gave protection up to 25 days.

Results of virus transmission experiments on plants treated with three different Systox concentrations applied as soil drenches are shown in Table 4. Even at the 1:200 concentration, where the aphids began to die 2 hours after being placed on the plants, the percentage of virus infection was fairly high. This infection was not surprising since it had been shown earlier that infective aphids could transmit the virus to healthy seedlings in as short a time as 5 minutes.

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RESULTS OF SEED TREATMENT TESTS FOR SMUT CONTROL IN NATURALLY INFESTED OATS AND ARTIFICIALLY INOCULATED OATS AND BARLEY[1]

Laurence H. Purdy[2]

Seed treatment tests in the East and South (3, 4) and Midwest (1) have demonstrated the effectiveness of many commercial fungicides in controlling loose smut of oats (Ustilago avenae (Pers.) Rostr.). Seed treatment tests with a few fungicides for the control of covered smut of barley (U. hordei (Pers.) Lagerh.) were reported by Porter in Colorado (7). A limited uniform cooperative seed treatment test for the control of oat and barley smuts has been conducted at Pullman, Washington, for several years (5). The results presented here are from tests designed to determine the effectiveness of several of the newer seed treatment fungicides in controlling oat and barley smuts in the Pacific Northwest.

MATERIALS AND METHODS

Anthony oats (C. I. 2143) and Trebi barley (C. I. 936) were used in these tests. In the first two experiments all of the oat seed was inoculated by the partial vacuum method. The seed was placed in a spore suspension, consisting of about 1 gram of spores per liter of water, and evacuated for 30 minutes, after which it was removed, dried, and treated. In the other tests both vacuum-inoculated and naturally infested oat seed was used. The seed of Trebi barley was inoculated by a modification of the method described by Tapke and Bever (8). The seed was soaked for 15 minutes in a spore suspension of 1 gram of spores per liter of water, then incubated for 24 hours at 20° C while still wet. The inoculated seed was then dried and treated.

Eighteen different chemical formulations were tested in one or more experiments over the 4-year period, 1954-1957. The materials tested, their active ingredients, and the manufacturers are listed below.

Agrox	6.7% phenylmercuriurea. Chipman Chemical Co., Bound Brook, New Jersey.
Ceresan M	7.7% N-(ethylmercuri)-p-toluenesulfonanilide. E. I. du Pont de Nemours & Co., Wilmington, Delaware.
Ceresan M-2X	15.4% N-(ethylmercuri)-p-toluenesulfonanilide. E. I. du Pont de Nemours & Co., Wilmington, Delaware.
Ceresan 75	2.80% ethylmercury 2,3-dihydroxypropyl-mercaptide and 0.60% ethylmercury acetate. E. I. du Pont de Nemours & Co., Wilmington, Delaware.
Ceresan 200	6.00% ethylmercury 2,3-dihydroxypropyl-mercaptide and 1.30% ethylmercury acetate. E. I. du Pont de Nemours & Co., Wilmington, Delaware.
Emmi	10.34% 1,4,5,6,7,7-hexachloro-N.-(ethylmercuri) bicyclo[2.2.1] hept-5 ene-2,3-dicarboximide. Velsicol Corp., Chicago, Illinois.

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[1] Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Agricultural Experiment Stations of Idaho, Oregon, and Washington.

[2] Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Pullman, Washington. Scientific Paper No. 1683, Washington Agricultural Experiment Station, Pullman, Washington.

Heptachlor + PMA (Cal. Spray)	20% heptachlor + 1.0% phenylmercury acetate. California Spray-Chemical Corp., Richmond, California.
Heptachlor + PMA (Penn. Salt)	20% heptachlor + 2.0% phenylmercury acetate. Pennsylvania Salt Manufacturing Co., Tacoma, Washington.
Heptachlor + PMA (Stauffer)	20.6% heptachlor + 1.03% phenylmercury acetate. Stauffer Chemical Co., North Portland, Oregon.
Liquid Mercury 244	2.24% phenylmercury acetate and 1.57% ethylmercury acetate. E. I. du Pont de Nemours & Co., Wilmington, Delaware.
Mema	11.4% 2-methoxyethylmercury acetate. Chipman Chemical Co., Bound Brook, New Jersey.
Mer-sol	7.0% phenylmercury acetate. Stauffer Chemical Co., North Portland, Oregon.
Ortho LM Seed Prectant	2.7% methylmercury 8-hydroxyquinolate. California Spray-Chemical Corp., Richmond, California.
Ortho LM + Heptachlor	0.51% methylmercury 8-hydroxyquinolate + 26.7% heptachlor. California Spray-Chemical Corp., Richmond, California.
Panogen 15	2.2% cyano (methylmercury) guanidine. Panogen Inc., Ringwood, Illinois.
Panogen 42	6.3% cyano (methylmercury) guanidine. Panogen Inc., Ringwood, Illinois.
Setrete	7.0% phenylmercury acetate. W. A. Cleary Corp., New Brunswick, New Jersey.
Terraclor	75% pentachloronitrobenzene. Olin Mathieson Chemical Corp., Baltimore, Maryland.

The wettable powders were applied to the seed as slurries. To apply wettable powders at the 1/2 ounce per bushel rate, a 120 mg sample of powder was placed in a vial containing 2 ml of water, and 2 to 4 drops of Methocel (5% methylcellulose) were added to serve as a spreader and sticker. The slurry was then poured into a one-liter Erlenmeyer flask containing 250 ml of seed. The fungicide was distributed uniformly over the seed by shaking and rotating the flask until all free moisture was removed from the inner wall of the flask. Higher rates of application were made by increasing the amount of fungicide in 2 ml of water.

The liquid fungicides that were applied at 1/2 ounce per bushel were diluted 1:16 with water, and 2 ml of this dilution were used to treat 250 ml of seed. Higher or lower rates of application were made by altering the dilution ratio. The fungicide-insecticide combinations were applied to the seed undiluted. For applications of 4 ounces per bushel 0.85 ml of the fungicide was used to treat 250 ml of seed.

The treated seed was stored in folded paper bags in the laboratory for 7 or more days and then transferred to seed envelopes prior to planting. One hundred seeds were planted in each 5-foot row in the field tests, while about 25 seeds were planted in an 18-inch row in greenhouse tests. There were three replications of each treatment. The field tests were located at Pullman, Washington, and Moscow, Idaho.

Table 1. Results from seed treatment tests for the control of loose smut in vacuum-inoculated oat seed in 1954 and 1955.

Treatment	Form	Rate (Ounces per bushel)	Percent smutted panicles: Greenhouse 1954	Field 1954	Field 1955
Check -- untreated			56	64	82
Agrox	W[a]	1/2	20	3	88
Mema	L[b]	1/4	3	.5	73
Panogen 15	L	3/4	0	.2	4
Panogen 42	L	1/5	0	0	5
Ceresan M	W	1/2	8	.5	25
Ceresan M-2X	W	1/4	0	.3	22
Setrete	L	1/2	7	8	47
Emmi	L	1/2	0	0	17
Mer-sol	L	1/2	20	6	83
Liquid Mercury 244	L	3/4	0	0	60

[a] Wettable powder.
[b] Liquid.

Table 2. Average percentages of smut produced in greenhouse and field tests for the control of loose smut in naturally infested and vacuum-inoculated oats in 1955, 1956, and 1957.

Treatment	Form	Rate (Ounces per bushel)	Percent smutted panicles: Naturally infested	Vacuum inoculated
Check -- untreated			26	89
Panogen 15	L[a]	3/4	1	63
Panogen 42	L	1/5	2	69
Ceresan 75	L	3/4	3	69
Ceresan 200	L	1/4	3	66
Ceresan M	W[b]	1/2	1	58
Mema	L	1/4	9	68
Emmi	L	1/2	2	66
Check			20	87
Agrox	W	1/2	14	89
Liquid Mercury 244	L	3/4	3	72
Check			28	83
Heptachlor + PMA (Cal. Spray)	L	4 1/2	3	74
Heptachlor + PMA (Penn. Salt)	L	4	.5	41
Heptachlor + PMA (Stauffer)	L	4 1/2	3	74
Terraclor	W	1	4	88
Ortho LM Seed Protectant	L	3/4	2	56

[a] Liquid.
[b] Wettable powder.

Table 3. Average percentages of smut in a test for the control of covered smut of barley by seed treatment, 1957.

Treatment	Form	Rate (Ounces per bushel)	Percent smut
Check			27
Panogen 15	L[a]	3/4	0.2
Panogen 42	L	1/5	0.3
Ceresan 75	L	3/4	1.0
Ceresan 200	L	1/4	0.0
Ceresan M	W[b]	1/2	1.0
Mema	L	1/4	0.0
Emmi	L	1/2	0.3
Terraclor	W	1	16.0
Heptachlor + PMA (Penn. Salt)	L	4	0.0
Heptachlor + PMA (Stauffer)	L	4 1/2	8.0
Ortho LM Seed Protectant	L	3/4	1.0
Ortho LM + Heptachlor	L	3 1/2	0.0

[a] Liquid.
[b] Wettable powder.

The effectiveness of the test materials was determined by counting the smutted and the total number of panicles or heads per row.

RESULTS AND CONCLUSIONS

The results of three tests for oat smut control on vacuum-inoculated seed are summarized in Table 1. One of these tests was made in the greenhouse and the other two in the field.

High percentages of smut, averaging from 56 to 82 percent, developed in the check rows of these experiments, providing a good test of the fungicides. Although the majority of the materials tested gave good control in both tests in 1954, only Panogen 15 and Panogen 42 were effective in 1955. The poor control obtained with Agrox, Ceresan M, and Mer-sol in the 1955 tests may be the result of vacuum inoculation, which makes control difficult, although these chemicals were fairly effective in 1954. In 1937 Leukel (2) pointed out that oat smut control by seed treatment was more difficult in vacuum-inoculated seed than in naturally infested seed. More recently Meiners (6) obtained similar results in seed treatment tests for smut control in vacuum-inoculated and naturally infested mountain brome grass. This would indicate that artificial inoculation by the vacuum method produces unnatural and unnecessarily difficult problems of smut control by seed treatment, even with the newly developed fungicides now available.

Tests with both naturally infested and vacuum-inoculated seed were made in the greenhouse in 1955 and 1956, and in the field in 1956 and 1957. The average results of these four tests are summarized in Table 2.

A fairly high incidence of smut (20 to 28 percent) developed in the untreated check rows in all tests with naturally infested seed, and good smut control was provided by all of the materials except Agrox and Mema. All of the formulations that provided good control are volatile mercurials, but fairly good control was obtained also with Terraclor (pentachloronitrobenzene) despite the fact that this material has low volatility.

High percentages of smut (83 to 89 percent) occurred in the untreated check rows of the tests made with vacuum-inoculated seed. None of the materials controlled smut in these tests. Obviously, inoculation of oat seed with smut spores by the vacuum method presents problems of smut control by seed treatment that are not encountered in naturally infested seed. Consequently, naturally infested seed would be preferable for evaluating seed-treatment fungicides for commercial use.

A test for control of barley covered smut by seed treatment was made in 1957, and the

results are presented in Table 3. A fairly high percentage of smut developed in the untreated check rows, the average being 27 percent. All treatments except two (Terraclor, and Heptachlor plus PMA [Stauffer]) provided good control. This relatively poor control of barley smut by Terraclor was in contrast to its fairly good control of oat smut. Laboratory tests on spores in the presence of Terraclor vapor showed that the germination of oat smut spores was inhibited, but this material had no effect on the germination of spores of barley smut.

While stand counts in the field tests generally showed no beneficial effect from the materials tested, some combinations of Heptachlor and PMA caused slight stand reductions.

From the data presented here it is evident that good control of oat or barley smut can be obtained with several of the standard mercury fungicides now on the market. The results also indicate that testing of new materials for oat smut control should be done with naturally infested seed since it more closely represents the conditions encountered in commercial treating than does artificially inoculated seed.

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AVENA ABYSSINICA SUSCEPTIBLE TO HELMINTHOSPORIUM BLIGHT[1]

H. R. Rosen and J. A. Spencer[2]

The finding of race 264 and other races of crown rust, Puccinia coronata var. avenae, that produce susceptible reactions on Landhafer and all other varieties used up to the present for incorporating resistance to crown rust, has centered attention on the need for parentage that offers resistance to these new races. Among the seven oat varieties or species which Dr. Marr D. Simons[3] listed recently as possessing resistance to these races was Avena abyssinica, C. I. 7233, an Iowa selection from P. I. 193958. This represents an oat found in Ethiopia (Abyssinia) according to D. J. Ward (6). T. R. Stanton (5, pp. 99-101) notes that A. abyssinica has 28 chromosomes (2 n) and this contrasts with common or most cultivated varieties with 42 chromosomes.

While C. I. 7233 was being grown in the greenhouse, along with others that were resistant to Landhafer races, for the purpose of crossing these with other oats, it was noted that approximately 50 percent of C. I. 7233 seedlings were blighting. The blades of these turned yellow at first, then straw-colored, and the crowns presented a water-soaked appearance. Most of these died within a week or two after the first symptoms were noted. When such dead or dying seedlings were placed in a moist chamber, they developed rather coarse mycelial growths which started sporulating within 5 days, the spores being readily recognizable as Helminthosporium with dark olivaceous walls and usually with one or more septa.

MYCOLOGICAL STUDIES

About 20 single spores were isolated and grown on PDA in petri dishes. Within a few hours they germinated from one or both ends, subsequent studies showing that such polar germination was typical. This is characteristic of Helminthosporium sativum and of its variety, H. sativum var. victoriae (Meehan & Murphy) Rosen, Wiser & York (4, p. 22).

Spore measurements of 100 or more spores derived from tube and plate cultures of PDA grown at 28° C were as follows: 23-58 x 8-15 microns, average 35 x 11 microns. In another series of measurements of 100 spores from plate cultures kept under very similar conditions, the following were obtained: 26-64 x 9-19 microns, average 43 x 12 microns. These measurements compare as follows with those obtained under similar conditions from cultures of the blight organism derived from blighted Victoria derivatives -- one culture obtained from S. S. Ivanoff measuring (200 spores): 15-53 x 8-15 microns, average 32 x 12 microns, and another series of cultures derived from a blighted Traveler plant (Victoria derivative) measuring (200 spores): 15-60 x 8-16 microns, average 31 x 13 microns.

So far as spore measurements are concerned, the organism derived from blighted Avena abyssinica plants cannot be distinguished from those derived from blighted Victoria derivatives. This also applies to spore color, shape, number of septa, thickness of exospore, and manner of germination.

PATHOLOGICAL STUDIES

Two methods of inoculation were used, first seed inoculation with mycelium and spores of pure cultures on PDA derived from single spores, the inoculated seeds kept at 28° C on seed-germinating paper moistened with fungal cultures diluted with water (Ivanoff's (1) "rag-doll" method); and second, seedling inoculation (2) in a greenhouse with mycelium and spores sprayed in water suspensions on the leaves and kept in a moist inoculating chamber for 48 hours after inoculation, the temperature in the greenhouse being held at 24° to 28° C.

Three different cultures were used, two isolated from Victoria derivatives mentioned above, and the other from Avena abyssinica. All three produced infections on Arlington, a Victoria derivative, with typical symptoms, and on A. abyssinica. The latter appeared to be just as susceptible to cultures obtained from Victoria derivatives as to those isolated from

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[1] Published with the approval of the Director, Arkansas Agricultural Experiment Station.

[2] Professor Emeritus and Research Assistant, respectively.

[3] In a circular letter addressed to plant breeders, pathologists and others, issued May 21, 1957.

blighted A. abyssinica plants. Furthermore, in the latter species, not only was the selection C. I. 7233 susceptible, but the original P. I. 193958[4] was equally so.

DISCUSSION

Since mycological and pathological studies showed no marked difference between the variously derived isolates, it is concluded that the blight on A. abyssinica is typical of the organism and of the disease that is commonly called "Victoria blight" or preferably "Helminthosporium blight" of oats.

In addition to the disadvantage of using A. abyssinica for breeding purposes occasioned by its chromosome count, its susceptibility to Helminthosporium blight makes it doubly undesirable.

Perhaps the main interest attached to the findings here reported resides in the fact that Avena abyssinica appears to have little or no botanical relationship to Victoria or its derivatives and has "no business" being susceptible to "Victoria blight". However, Rosen has previously called attention to the susceptibility of other oats that have no Victoria heritage (3).

Victoria is considered by Stanton (5, p. 90) as belonging to A. byzantina, the cultigens of A. sterilis, although he notes that the florets in Victoria separate by disarticulation as they do in varieties of A. sativa. Therefore, he suggests that Victoria may have resulted from hybridization of the two species. Whatever the origin of Victoria may have been, it is so different cytologically and morphologically from A. abyssinica that it is difficult to conceive of any close relationship between the two.

The conclusion is that the Helminthosporium blight pathogen has a considerably wider host range among oats than has been assumed and that the reduction of the species name H. victoriae to H. sativum var. victoriae gains in validity, since it more clearly indicates botanical relationship to a cosmopolitan species of relatively wide distribution and host range.

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[4] Seed kindly furnished by Marr D. Simons.

RHIZOCTONIA SOLANI MAY BE TRANSMITTED WITH SEED OF AGROSTIS TENUIS

Charles M. Leach and Merle Pierpoint

A rapid damping-off of Oregon-grown bentgrass seedlings (Agrostis tenuis) was observed in a few seed laboratory germination tests in 1954 and 1955. Symptoms during germination on illuminated moist filter papers were the formation of white web-like mycelia around the caryopses and coleoptiles, followed by rapid damping-off of the seedlings. In addition, minute brown sclerotium-like protuberances were produced on the surface of or under the glumes of some but not all infected seeds. Rhizoctonia solani Kuehn was consistently isolated. This is apparently the first record of the seed transmission of R. solani with seed of Agrostis tenuis.

To discover whether Rhizoctonia was internally or externally seed-borne, untreated and surface-disinfected seeds, taken from a sample known to be infested with the fungus, were placed on 2% malt extract agar and incubated at 21° C for 10 days. The seeds to be surface-disinfected were immersed in a mixture of sodium hypochlorite and ethanol[1] in a partial vacuum. Rhizoctonia infested 10.9 percent of the 568 untreated seeds and 1.7 percent of the 508 disinfected seeds.

Because of Oregon's importance as grass seed producer, the incidence of R. solani associated with bent grass seed passing through the Oregon State Co-operative Seed Testing Laboratory was determined. All samples undergoing routine germination tests that showed seedling symptoms similar to Rhizoctonia infection were noted by seed analysts. Of a total of 609 samples of seed tested, 111 showed symptoms that were believed to be due to R. solani. From these 111 samples 40 were randomly selected and germinated on blotters using 400 seeds per test. As the symptoms of Rhizoctonia infection in germination tests can be confused with those caused by other fungi, isolations were made from damped-off plants for accurate diagnoses. Rhizoctonia was not isolated from any of the retested samples. Fungi isolated were Botrytis cinerea, Fusarium sp., Epicoccum sp., Alternaria tenuis, Cladosporium sp., and an Ophiostoma sp.

Conclusions drawn from this study were, 1) though R. solani was proven to be transmitted with seed of Agrostis tenuis, the incidence of seed-borne Rhizoctonia in the Oregon-grown seed examined is negligible, and 2) detection of R. solani during routine blotter germination tests from seedling symptoms alone is unreliable.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, AND OREGON STATE COOPERATIVE SEED TESTING LABORATORY, CORVALLIS, OREGON

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ERADICATION OF RING ROT BACTERIA FROM CONTAMINATED POTATO BAGS BY MOIST HEAT TREATMENT[1]

L. T. Richardson and C. T. Buckland[2]

Summary

The thermal death rate of potato ring rot bacteria on infested jute fibers treated in air was found to vary with the moisture content of the air. The effects of three different humidity levels in air at 70°C on the temperature and on the survival of the organism at various locations within a bale of jute bags were determined. The rate of heating of the jute and the mortality rate of the bacteria at each location was found to vary with the moisture content of the air. The mortality rate at each humidity level was highest at the center of the bale, decreased towards the surface and was lowest in the free space outside despite the reverse temperature gradient. This apparent anomaly is attributed to the original moisture content of the jute.

INTRODUCTION

Corynebacterium sepedonicum (Spiek. & Kott.) Skapt. & Burkh., the organism causing bacterial ring rot of potato, has been shown to survive on dry jute for many months at normal storage temperatures and to be very resistant to dry heat[3]. An exposure to dry air at 150°C was found necessary to completely disinfest contaminated jute fibers in 10 minutes. Since large quantities of bags, particularly in bales, cannot be uniformly heated to such a temperature within a reasonable period of time, a study was made to determine the rate of kill at lower air temperatures and the effect on it of the relative humidity of the air.

METHODS

The effect of relative humidity on the viability of ring rot bacteria on jute was demonstrated first in a preliminary test where 2-inch jute fibers infested with pure cultures of the organism were exposed for various periods of time to a series of temperatures in dry and moist air. Samples enclosed in sterile petri dishes were heated to the required temperatures in small electric ovens with and without pans of water on the bottom shelf. At intervals samples were removed and transferred to tubes of liquid medium. After incubation the number of viable bacteria on each fiber was determined from turbidity readings following the method developed by the senior author[3].

Full-scale tests were next conducted with tightly wired bales comprising 300 jute bags heated in a large electric oven equipped with a circulating fan. Infested fibers enclosed in steel capsules with narrow slits to permit air circulation were inserted into the bale at depths of 4, 6, and 10 inches and also suspended in the free space above the top of the bale. Thermistor probes located in the same positions were connected with a meter outside the cabinet[4]. Wet- and dry-bulb thermometers were mounted through a rubber stopper in the top of the oven, the wick of the wet bulb being immersed in a tube of water within the cabinet.

In each of three tests made the temperature in the free space was maintained at approximately 70°C for 72 hours, then the bale was left in the closed oven with the heater and fan off for 48 hours. Three different relative humidity levels in the air surrounding the bales were achieved by varying the amount of free water present in the oven. In the first run no

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[1]Contribution No. 125 from the Science Service Laboratory, Canada Department of Agriculture, London, Ontario.

[2]Senior Plant Pathologist and Technician (Fumigation Unit), respectively. The authors are indebted to R. A. Ludwig and H. A. U. Monro for their interest and guidance in this investigation.

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[4]"Tele-Thermometer" manufactured by Yellow Springs Instrument Co., Yellow Springs, Ohio.

water was provided, in the second pans of water were placed on shelves above and below the bale, and in the last run a greater volume and surface area of water was provided and the door was tightly sealed with tape to reduce moisture loss.

Temperature readings for all positions were recorded at least every 3 hours during the heating and cooling periods. Samples were removed for viability determinations at 24-hour intervals. Each bale was weighed before and after treatment to determine the amount of moisture lost.

RESULTS

The results of the preliminary tests with infested fibers exposed to dry and moist heat at various temperatures indicated that the rate of loss of viability of the organism is increased when the relative humidity of the air is increased. An example is shown in Table 1 which lists the average number of survivors per fiber following a series of exposures to air at 60°C and 10 and 55 percent relative humidity.

Table 1. Effect of humidity on the viability of ring rot bacteria on jute fibers exposed to 60°C.

Exposure time (Hr.)	Number of viable bacteria[a]	
	R.H. 10%	R.H. 55%
0	266,000,000	266,000,000
2	-	132,000,000
4	125,200,000	7,500,000
8	56,200,000	1,700,000
16	204,000,000	2,080,000
24	176,000,000	507,000
48	14,000,000	105,000
72	14,700,000	-
76	4,100,000	-

[a]Average of 5 replicates

The relative humidity of the air and the temperatures at the various locations over the course of the three full-scale tests are represented in Figures 1 to 3. In the absence of free water the relative humidity declined from 17 percent to 7 percent in the first run. The temperature within the bale increased gradually, reaching 64° and 53°C at the 4- and 6-inch depths by the end of the heating period. At the center of the bale, however, the temperature reached 47° in 35 hours but increased no further. In the second run (Fig. 2) the relative humidity was maintained at approximately 22 percent except when the pans went dry. The internal temperatures rose more rapidly than in the previous test and increased further each time the water was replenished. The humidity in the final run (Fig. 3) was 42 percent while water was present. The internal temperatures rose very sharply at the beginning and again at 48 hours when the water was replenished. The temperature differences between locations within the bale were less when the relative humidity of the air was maintained at a high level than when it was low.

The effect of the relative humidity of the air on the amount of moisture lost from the jute is apparent from the initial and final weights of the bales listed in Table 2.

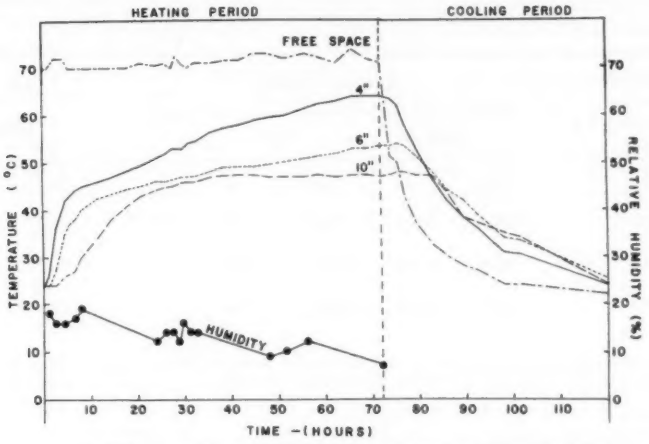

FIGURE 1. Temperature and relative humidity in free space and temperatures at three different depths within a bale of jute bags heated for 72 hours in oven with no water provided.

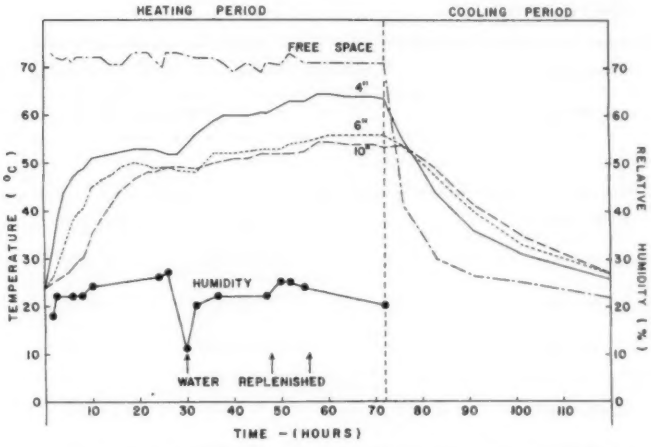

FIGURE 2. Temperature and relative humidity in free space and temperatures at three different depths within a bale of jute bags heated for 72 hours with pans of water in oven.

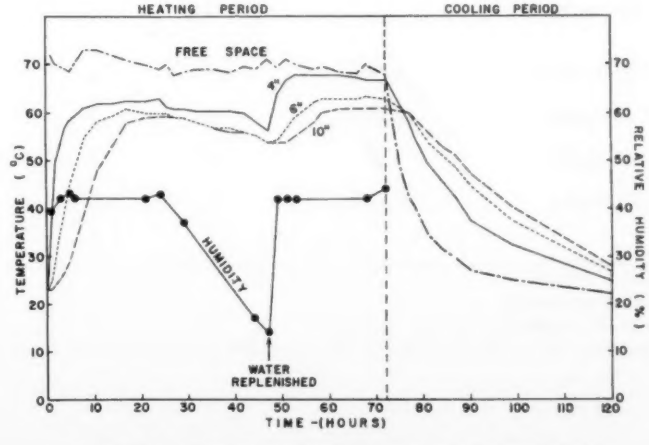

FIGURE 3. Temperature and relative humidity in free space and temperatures at three different depths within a bale of jute bags heated for 72 hours with pans of water in oven and door sealed.

Table 2. Effect of relative humidity of air at 70° C on the amount of moisture lost from bales of jute bags in 72 hours.

Relative humidity	Moisture Content[a]				Net loss	
	Initial		Final			
%	lb.	%	lb.	%	lb.	%
7-17	20.8	13.8	9.8	6.5	11.0	7.3
22	20.9	13.9	12.2	8.1	8.7	5.8
42	15.3	10.0	12.8	8.4	2.5	1.6

[a]Percentages calculated on dry weight basis.

Table 3. Effect of heating bales of jute bags for 72 hours in air at 70° C and three different humidity levels on the viability of ring rot bacteria on infested jute fibers located at various positions within each bale and in the free space above it.

Relative humidity (%)	Position of sample	Average number of viable bacteria per sample[a]			
		Treatment time (hours)			
		24	48	72	120
7-10	Free space	—	2,268,000	2,034,000	1,314,560
	4" depth	—	2,012,700	722,000	5,750,000
	6" depth	—	10,000	700	236,000
	10" depth	—	20,200,000	2,250	15,500
22	Free space	3,200,000	50,000	337,500	212,250
	4" depth	—	1,500	1,460	50,400
	6" depth	—	25,700	3	18
	10" depth	—	36,800	<1	0
42	Free space	1,300	677	<1	<1
	4" depth	1,015	0	X	<1
	6" depth	X[b]	<1[c]	X	0
	10" depth	150,000	<1	<1	0

[a]Average of 5 replicates
[b]Cultures contaminated
[c]One to four of replicates with 1 survivor
N. B.: Population of untreated fibers 161, 800, 000 (average of 29 replicates)

Table 3 is a summary of the average number of viable bacteria found on samples from the various locations at different periods in the course of the three heat treatments under different humidity conditions. Survival in all positions was influenced by the humidity of the outside air. When no free water was provided complete kill was not achieved at any position by the end of the cooling period. With 22 percent moisture in the air virtually complete kill was attained at the 6- and 10-inch depths within 72 hours but not at the 4-inch depth or the free space by the end of the cooling period. With 42 percent humidity there was no appreciable survival at any position in the bale at 48 hours and very little in the free space. Contrary to expectations from the temperature data, survival in each case was least at the center of the bale (except at the first sampling time), increased towards the surface of the bale, and was greatest in the free space outside the bale.

DISCUSSION

The results of these tests clearly indicate that the relative humidity of the air is an important factor governing the effectiveness of treatments to eradicate ring rot bacteria from contaminated jute bags. It has a direct effect on the thermal death rate of the organism as demonstrated in the preliminary tests. High external humidity also affects the rate of heating in the interior of a bale of bags, presumably by improving the penetration of heat and reducing the amount of heat lost through evaporation. The increased mortality rate at all positions with increased external humidity is probably the result of its effect on both temperature and humidity in the interior of a bale. The fact that the mortality rate in all three trials was highest at the center of the bale and lowest outside despite the reverse temperature gradient appears anomalous but can attributed to high humidity conditions within the bale provided by the original moisture content of the jute. The higher the external humidity the better this internal humidity will be maintained and the shorter will be the time required for complete mortality.

Specific recommendations as to the number of hours required to maintain jute bags at a given temperature in order to achieve complete eradication in practice cannot be made on the basis of the results of these tests. Since conditions will vary with the facilities available, the volume, compactness and moisture content of the jute bags to be treated, and other factors, the effectiveness of heat treatments will need to be evaluated under each individual set of conditions. For the most effective treatment in the shortest possible time the moisture content of the jute should be relatively high and the humidity of the air maintained at the highest possible level by whatever means are available.

SCIENCE SERVICE LABORATORY, CANADA DEPARTMENT OF AGRICULTURE

EARLY BLIGHT OF POTATOES IN SOUTHEASTERN IDAHO

James W. Guthrie[1]

Early blight of potatoes, caused by Alternaria solani (Ell. & G. Martin) Sor., although well known in many areas of the United States, has not been important in southeastern Idaho until recent years. This disease is, however, not unknown to Idaho. Under the proper conditions, the target spots characteristic of this disease have appeared on the foliage in many fields irrigated by the gravity system, but only in trace amounts.

During the past 5 years, with the development of new land not suitable for the gravity method of irrigation, sprinkler irrigation has come into wider use. Following the advent of sprinkler irrigation early blight began to appear in scattered areas, varying from a trace to 50 percent defoliation. During 1957 complete defoliation of the plants occurred in many fields, which involved about 3000 acres. This includes areas north and west of Blackfoot, Idaho as well as the Minidoka project north of Rupert, Idaho. In every instance the fields were irrigated by sprinklers.

Frequent rains or heavy dews during periods of high temperature are essential for abundant sporulation and to provide water for germination of conidia. The sprinkler system of irrigation also appears capable of providing the moisture conditions for a severe epiphytotic. This is particularly true during late July and early August, when the crop is frequently irrigated at 5- to 7-day intervals. So far the destructiveness of this disease has been confined to defoliation. No confirmed cases of tuber rot have been observed.

In addition to providing conditions favorable for the development of the disease, sprinkler irrigation also complicates control measures. During July and August, which is a critical period of plant growth, water often may be applied at intervals of 5 days. This means that unless an adequate fungicide can be made to stick to the leaves the foliage must be sprayed or dusted following each irrigation. So far no attempt has been made to control this disease, but preliminary testing of fungicides is planned for the coming year.

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A PRELIMINARY REPORT ON THE CONTROL OF DECAY OF PORTO RICO SWEETPOTATOES DURING MARKETING

L. J. Kushman and G. B. Ramsey

INTRODUCTION

Losses from Rhizopus rot occur in sweetpotato roots after they are removed from storage, washed, graded, and shipped to market (4, 5, 7, 10). Since the decay-producing organisms enter injuries made during preparation for shipment, it has been suggested that roots be recured after grading so that injuries will heal just as at harvest (2). Some success has been reported in controlling decay due to Rhizopus when washed and graded roots were immediately recured or transported short distances and then recured before restorage (1).

Another approach to the control of decay during marketing of sweetpotatoes involves chemical treatments. Dipping roots in 1% to 2% borax solutions provided some control of soft rot (1, 3) and black rot (8). A wax emulsion containing Dowicide A (97% sodium orthophenylphenate) was ineffective in the control of black rot of sweetpotatoes, but it was not evaluated for soft rot control (8). Since then it has been found that Rhizopus decay of peaches was reduced by Dowicide A treatment (9).

The authors have made several curing or recuring tests to date but have obtained the most promising results with certain chemical washes applied to sweetpotatoes after the roots were removed from storage and prepared for shipment. The data are reported herein.

METHODS

In preliminary holding tests several chemicals including 2-aminopyridine, captan, sorbic acid, Vancide 51, and diphenylamine were tried, but none controlled decay as well as borax or Dowicide A. Therefore further tests were made with the last two chemicals.

For holding tests sweetpotato roots that had been cured and stored for a month or more were injured by tapping one end of the root with a solid object so that a shattered bruise resulted. The injured roots were then dipped momentarily in the test solution, drained, and placed in paper bags at 60° F for 10 days. In each test, ten roots were used in each of four replicates per treatment. The amount of decay was recorded at the end of the 10-day holding period as a percentage of the number of roots.

Roots injured and treated in much the same manner were used in two shipping tests from Sunset, Louisiana, to Chicago, Illinois. A 1-bushel crate of sweetpotatoes was used per treatment.

In three additional shipments, roots injured only in the normal washing, grading, and handling process were treated after grading and shipped. Two 1-bushel crates were used per treatment.

The test crates were placed in commercial shipments carried by vans during the winter. A Ryan recording thermometer included in one crate in each shipment showed transit temperatures between 50° and 65° F for the 36- to 48-hour transit periods. Decay (mostly due to Rhizopus) on arrival in Chicago and after holding 1 week at 70° to 75° was recorded, but only the data obtained after holding 1 week are reported.

A small amount of Nacconol was added to all solutions as a spreader. Since Dowicide A produced chemical injury in the first tests, hexamine, at 50 percent the concentration of Dowicide A, was added as a safener in later tests (6). In one test treated roots were rinsed with water by passing them under a water spray nozzle mounted over a conveyor belt on the grading equipment. The sweetpotato variety used in all tests was Porto Rico.

RESULTS AND DISCUSSION

Dowicide A and borax reduced or eliminated decay in holding tests covering a fairly wide range of concentrations (Table 1). The results of similar tests were averaged because of the consistency of the data. The results of the holding tests were similar to those of the shipping tests. Borax, at 1%, reduced decay in all comparisons, but, on the average, not as much as Dowicide A at 5% or 1%. Even in dilute solutions Dowicide A caused chemical injury unless hexamine was added. A water rinse after treatment with Dowicide A did not adversely affect decay control.

Table 1. Decay of Porto Rico sweetpotatoes treated with Dowicide A or borax before holding or shipping.

Chemical name and concentration (percent)	Roots in holding test: Number tested	Roots in holding test: Percent decayed[a]	Roots in shipping tests: Percent injured roots decayed[b]	Roots in shipping tests: Percent graded roots decayed[b]
None	240	76	61	5
Dowicide A:				
.10	80	21	62	-
.25	80	7	24	-
.50	240	3	2	1
1.00	240	1	0	0
Borax:				
.25	80	10	38	-
.50	80	3	14	-
1.00	240	5	10	2
2.00	80	0	-	-
4.00	80	0	-	-

[a] Based on number of roots after holding 10 days at 60°F.

[b] Based on weight of roots shipped after holding 1 week in the market.

The results of the tests with Dowicide A and borax have been sufficiently encouraging to warrant additional tests. The use of a chemical dip to control decay would change present methods very little and would require very little more labor or expense. However, neither Dowicide A nor borax has been approved for use on sweetpotatoes by the Food and Drug Administration (11).

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DISEASES OF WESTERN-GROWN LETTUCE ON THE PITTSBURGH, PENNSYLVANIA MARKET

B. A. Friedman

Ordinarily the amount of disease in all shipments of produce cannot be accurately estimated from Federal certificates inasmuch as inspections usually are not requested unless a shipment is damaged, with the result that often only a small percentage of shipments is inspected. However, during the period from 1935 to 1942 inspections were made on 24,737 carlots, or approximately 52 percent of all rail shipments of lettuce unloaded at New York City. In that study it was reported that 68 percent of the shipments showed decay, averaging 16.8 percent in affected cars or 11.5 percent decay in all shipments inspected. The principal decays reported were tipburn [nonparasitic], bacterial soft rot [Erwinia carotovora], gray mold not [Botrytis cinerea], and watery soft rot [Sclerotinia sclerotiorum].

The purpose of the present note is to report the results of a compilation of 726 inspections, or approximately 76 percent of all rail shipments unloaded at Pittsburgh, Pennsylvania, during the period from January to June, 1957[2]. The results are given in Table 1. Only 6.2 percent of the carlot shipments were reported free of disease. Decay was found in 83.3 percent of the inspections, and averaged 6.7 percent in affected cars or 5.6 percent in all cars. Tipburn was reported in 55.8 percent of the cars, averaging 16.7 percent for affected cars or 9.3 percent in all cars. Russet spotting, rib discoloration, and pink rib, formerly called the brown spot complex[3], were present in appreciable but smaller amounts.

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[1] Wiant, J. S. and C. O. Bratley. 1948. Spoilage of fresh fruits and vegetables in rail shipments unloaded at New York City 1935-42. U. S. Dept. Agr. Circ. 773, 62 pp.

[2] Acknowledgment is made to the Fruit and Vegetable Division, Agricultural Marketing Service, for permission to examine the inspection certificates.

[3] Friedman, B. A. 1954. Brown spot complex of head lettuce on eastern markets. Plant Disease Reptr. 38: 847-851.

Table 1. Diseases of lettuce in rail shipments unloaded at Pittsburgh, Pennsylvania, January-June 1957.

STATE OF ORIGIN [a]		ARIZONA						CALIFORNIA							COLORADO			N.M.	TEXAS					ALL
MONTH [b]		J	F	M	A	M	S.T.	J	F	M	A	M	Ju.	S.T.	A	M	S.T.		J	F	A	M	S.T.	T.
TOTAL CARLOTS INSPECTED	No.	9	21	55	96	23	204	26	105	88	57	159	22	457	14	11	25	14	3	18	2	3	26	726
CARLOTS HAVING NO DEFECTS	%	44	14	4	4	4	6.9	15	17	2	2	1	0	5.9	0	18	8.0	0	0	11	0	0	7.7	6.2
DECAY [c]																								
CARS AFFECTED	%	11	52	95	88	83	81.9	44	57	92	95	99	100	84.2	79	91	84.0	92.9	67	67	100	100	73.1	83.3
AMOUNT IN AFFECTED CARS	%	3	3	9	5	5	5.9	1	2	6	8	8	13	6.9	9	13	10.9	4.9	5	2	2	21	5.4	6.7
AMOUNT IN ALL CARS	%	<1	2	8	4	4	4.9	1	1	5	8	8	13	5.8	7	12	9.1	4.5	3	2	2	21	4.0	5.6
TIPBURN																								
CARS AFFECTED	%	22	29	71	67	96	65.2	8	37	49	77	52	50	48.4	79	91	84.0	92.9	67	61	50	100	65.4	55.8
AMOUNT IN AFFECTED CARS	%	7	5	15	9	24	17.3	10	7	21	21	16	13	15.9	17	31	23.5	28.6	7	5	2	5	5.3	16.7
AMOUNT IN ALL CARS	%	1	1	11	6	23	11.3	1	2	10	16	8	7	7.7	13	28	19.7	26.6	5	3	1	5	3.5	9.3
RUSSET SPOTTING																								
CARS AFFECTED	%	0	0	22	<1	0	10.8	0	0	6	12	2	5	3.5	14	0	8.0	0	0	0	0	0	0	5.5
AMOUNT IN AFFECTED CARS	%	0	0	22	9	0	15.8	0	0	13	16	6	5	12.6	10	0	9.5	0	0	0	0	0	0	14.2
AMOUNT IN ALL CARS	%	0	0	5	1	0	1.7	0	0	73	20	<1	<1	0.4	1	0	0.7	0	0	0	0	0	0	0.8
RIB DISCOLORATION																								
CARS AFFECTED	%	0	14	6	<1	4	8.8	0	2	9	4	4	0	3.5	14	0	8.0	7.1	67	22	0	33	26.9	6.1
AMOUNT IN AFFECTED CARS	%	0	3	7	8	4	6.6	0	4	6	2	4	0	4.4	2	0	2.0	0.2	6	20	0	5	13.7	6.6
AMOUNT IN ALL CARS	%	0	<1	<1	1	<1	0.6	0	<1	<1	<1	2	0	0.3	<1	0	0.2	2.5	4	4	0	2	3.7	0.4
PINK RIB																								
CARS AFFECTED	%	56	48	18	16	0	19.6	52	24	9	11	23	46	21.0	29	0	16.0	7.1	33	11	50	67	23	20.2
AMOUNT IN AFFECTED CARS	%	10	6	9	7	0	7.6	7	7	4	5	8	10	7.2	4	0	4.0	9.0	3	8	5	6	5.8	7.2
AMOUNT IN ALL CARS	%	6	3	2	1	0	1.5	4	2	<1	1	2	8	1.5	1	0	0.6	1.2	1	1	3	4	1.3	1.5

[a] N. M. equals New Mexico.

[b] Month: J, F, M, A, M, Ju refer to January, February, March, April, May and June respectively. S. T. equals subtotal; T. equals total.

[c] Decay was reported to be almost entirely bacterial soft rot.

THE DEVELOPMENT OF AN EFFICIENT SCHEDULE FOR THE USE OF KARATHANE IN THE CONTROL OF APPLE POWDERY MILDEW[1]

A. B. Groves, E. L. Wampler and C. B. Lyon[2]

Summary

Three experiments essentially identical in design were conducted in Virginia and California to evaluate the influence of the rate of usage and time intervals between sprays on the fungicidal effectiveness of Karathane for the control of apple powdery mildew. Although both factors were of substantial importance, the time factor emerged as the most important by a ratio of approximately four to one.

The conclusion drawn from these experiments is that the most efficient way to utilize Karathane for mildew control is to apply small amounts at frequent intervals. A usage rate of 8 ounces at not over 2-week intervals is suggested as feasible for general cover spray practice. Modifications early in the season in favor of a shorter time interval and lower rate of usage may also prove satisfactory, and would better fit the early season scab and rust protective schedules necessary in many of the apple growing regions.

Apple powdery mildew (Podosphaera leucotricha) has been a disease problem of major importance for many years in Western United States orchards on such susceptible varieties as Jonathan, Gravenstein, and Rome. The early use of sulfur was not a common practice in these regions, as it was in the eastern apple-producing regions where sulfur was used for scab control. Sulfur was employed for the control of mildew as the only available effective mildew fungicide, but usage was restricted by phytotoxic considerations.

A rapid increase in the occurrence and severity of powdery mildew in the East followed a widespread shift away from sulfur to the organic fungicides, principally captan (N-(trichloromethylthio)-4-cyclohexene-1,2-dicarboximide), ferbam (ferric dimethyldithiocarbamate), glyodin (2-heptadecyl-2-imidazoline acetate), and the phenyl-mercury fungicide (tris (2-hydroxyethyl) (phenylmercuri) ammonium lactate and phenylmercury acetate). None of these fungicides are effective in the control of powdery mildew. The mildew problem, which had formerly been of minor importance, became a major problem on susceptible varieties.

A return to the use of sulfur would not be a wholly satisfactory solution because of the limitations imposed by phytotoxic and physiological responses associated with its use on some varieties, and by the normally high day temperatures encountered in many apple-growing regions during the time protection against powdery mildew is necessary.

The normal infection period extends from an early preblossom stage of bud development until approximately a week after terminal growth has ceased. New infections continue to occur so long as new and susceptible foliage is being developed. The period of susceptibility to mildew infection in a normal season is usually from 8 to 12 weeks.

Sulfur cannot be safely used throughout the infection period in those areas where the disease is a problem. Karathane (2-(1-methylheptyl)-4,6-dinitrophenyl crotonate and isomers) is the only other currently available fungicide that is effective against the powdery mildew fungus. The karathane formulation commercially available and used in these experiments is a 25% active ingredient, dry wettable powder. The rates of usage given are in terms of formulated product, not of active ingredient.

Experience in the use of Karathane was limited at the time the need for control measures became apparent. Early attempts to introduce the material into the spray programs of various regions met with varying degrees of success. Optimum rates of usage and the relation-

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[2]Plant Pathologist, Virginia Agricultural Experiment Station, and research personnel, Research Division Rohm and Haas Company, respectively.

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ship of timing intervals between fungicide applications were uncertain. The influence of air temperatures and rates of usage on the phytotoxicity of Karathane had not been established. Also unknown was the influence of chemical purity of the active ingredient, of formulation, and of surfactant additive.

The purpose of these investigations was to develop information on the use of Karathane, and its formulation, relevant to these factors. The determination of the most efficient and safe manner for the use of the material was the ultimate objective.

1956 VIRGINIA EXPERIMENTS

The experiments conducted in 1956 were designed to test three formulations of Karathane and two surfactants. Methods for the accurate determination of disease control were developed during these trials and were employed throughout the course of these experiments.

Orchard plots consisted of four trees each, replicated three times and randomized. All applications were made as dilute sprays with a high pressure spray rig at 600 pounds pressure with an 8-nozzle broom by a single operator on the rig. Each tree was sprayed to completion from two sides before proceeding to the next tree. Eight-year-old Rome trees with a recent history of severe powdery mildew infection were selected for the test.

The applications of Karathane and surfactants were made in simultaneous combination with the fungicides and insecticides as recommended in the 1956 Virginia Spray Bulletin. Spray applications began at the pre-pink stage of bud development on April 20, and subsequent applications were made on May 1, 7, and 22, June 4 and 19, and July 2 and 16.

Preliminary foliage mildew infection counts were made in late May. Final counts were made in mid-July. A determination of the incidence of mildew was based on a count of actual number of infections per leaf on the foliage of terminal growth.

The method used in making infection counts is described. Four terminal shoots were selected at random from each of four directional quadrants of each tree, and the number of infections per leaf recorded in sequence from the tip of the terminal inward. The count was begun with the fourth, or first fully expanded, leaf in the preliminary count and five leaves were scored. Scoring was begun with the terminal leaf and included ten leaves in the final count. This method was used without change in Virginia in both 1956 and 1957. Analyses of variance established the precision of the method and indicated that the sampling procedure was adequate and obtained the desired results.

Leaves having more than five lesions per leaf were recorded as five because of the difficulty of distinguishing greater numbers due to coalescing of lesions. Treatments which permitted such high rates of infection were considered to have failed, and the relative degree of failure to be of no importance. The grouping of higher numbers of lesions than five into the five classification doubtless distorts or minimizes the magnitude of difference between treatments vs. untreated. The scoring method does, however, cover the range of satisfactory treatments.

Three experimental Karathane formulations and two surfactants were tested in 1956. Surfactants included Triton B-1956 (a modified phthalic glycerol alkyd resin) and Triton X-114 (an alkylated aryl polyether alcohol).

All Karathane formulations were initially used at 1 pound per 100 gallons of spray, and the standard formulation was also used at a 1 1/2-pound rate. Both surfactants were used at a 2-ounce rate.

The preliminary (late May) count revealed differences between formulations to be not demonstrable as yet and decision was made to reduce the dosage of Karathane by half on two of the four trees for the remainder of the season. It was hoped that this would result in a segregation of biological performance between the combinations.

After the final count it was determined that under the conditions of this experiment each of the Karathane formulations afforded essentially equal control of powdery mildew. Slight differences observed between the formulations were, on the average, within the limits of experimental error. Main effect comparisons revealed that over the range of the six formulations employed, halving the dosage for the four sprays did result in a statistically significant reduction in mildew control. The difference between the control afforded by full and half dosage was 97.4 vs. 95.5 percent. This small difference is considered to be of doubtful practical significance.

Comparisons made between four rate-of-usage levels (8, 12, 16, and 24 ounces per 100 gallons) of regular Karathane plus Triton X-114 surfactant revealed statistically significant (5 percent level) differences in the mildew counts. The differences were very small and are

of doubtful practical significance. Expressed in terms of percentage control, the range was 94 percent control at the 8-ounce rate to 98.7 percent for the 24-ounce rate.

These results led to the design of an extensive experimental program to examine the effects of rate of usage and the time interval between sprays on the biological performance of Karathane. These experiments were conducted in Virginia and California in 1957.

1957 EXPERIMENTS

Three experimental spray programs of essentially identical design were developed for 1957. The purpose of all three was to determine the most efficient manner of usage of Karathane through an evaluation of the influence of rate of usage and timing intervals on disease control. Two of the experiments were located in California and one in Virginia. It was hoped that this placement of tests in regions of widely differing climatic conditions would reveal the possible general applicability of the results.

Regular production Karathane, wettable powder formulation, was utilized in a factorial design involving four rates of usage (4, 8, 12 and 16 ounces per 100 gallons) and three timing intervals (7, 14, and 21 days) in both California and Virginia. These factors were constant in all three tests. There were deviations from uniform procedure in each of the situations, however. The three experiments are outlined and discussed separately to identify these differences.

Experiment No. 1 -- O'Brien Orchard, Berryville, Virginia

Nine-year-old Rome trees with a recent history of moderate to severe mildew infection were utilized. The entire block, including controls, was sprayed uniformly with a captan fungicide and appropriate insecticides throughout the season in conformity with the Virginia Spray Service recommendation. Karathane applications began in the early petal-fall stage of bud development and continued for 9 weeks, or through the time for the fourth cover spray. Triton B-1956 surfactant was added to all Karathane sprays at 2 fluid ounces per 100 gallons of spray. All Karathane sprays were made separately from the regular spray program mentioned earlier. The trees scheduled to receive weekly treatments were sprayed nine times; those sprayed every 2 weeks were sprayed five times; those sprayed every 3 weeks received three applications. All treatments began on the same date. The captan-insecticide applications were made separately and usually on other dates. All sprays were applied with the same rig and in the same manner as in 1956.

The 1957 season in Virginia was highly favorable for mildew development. The early season was abnormally warm and dry and infection began early. Powdery mildew control was evaluated only twice during the season.

Readings of foliage injury resulting from Karathane were taken following a period of abnormally hot, dry weather in June when it was apparent that some underleaf bronzing had occurred. The data are summarized in Table 4. Injury developed only on the weekly treatments which were sprayed during the period of hot weather. None of the treatments developed any visible injury where the residues were 1 or 2 weeks old. The injury produced by the two lower rates of usage was not considered to be of practical importance.

Experiment No. 2 -- Barnes Manzanita Ranch, Julian, California

Large mature Rome apple trees were used in this experiment. The orchard had a long history of moderate powdery mildew infection. The treatments were applied with a conventional orchard sprayer using hard guns at a pump pressure of 500 pounds. The trees were sprayed to full orchard coverage using an average of 16 gallons per tree. Applications were begun on May 24 and continued at the specified intervals through July 4. The trees scheduled to receive weekly treatments received six sprays; those treated every 2 weeks received three sprays, and the trees sprayed at 3-week intervals received two applications. All treatments included 2 pounds of dry wettable 50% DDT in a single application in early June. No other insecticides or fungicides were employed. Single tree plots were used and were replicated six times.

Mildew ratings were made by random selection of 30 mature leaves from the periphery of the tree at shoulder height while completely circling the tree. The percentage of leaf area infected by mildew was estimated on each leaf, thus combining both number of leaves infected and severity of infection. Numerical ratings on severity (area) of infection were: 0 = no in-

fection, 1 = 1 to 5 percent, 2 = 6 to 11 percent, 3 = 12 to 25 percent, 4 = 26 to 50 percent, and 5 = 51 to 100 percent, of leaf area infected.

The degree of powdery mildew control achieved was evaluated eight times, at weekly intervals beginning on May 28 and continuing through July 18.

Experiment No. 3 -- W. L. Farmer Ranch, Julian, California

Ten-year-old red sport Jonathan trees were used in this experiment. The method of spray application was the same as in Experiment 2. Spray applications were begun on April 12 and continued through August 8 for a total of 14 sprays at weekly intervals. Single tree plots were used, randomized and replicated four times.

The initial Karathane spray was applied after a severe infection was well established. Subsequent weather conditions were very favorable for heavy mildew development. For these reasons, results from this experiment may be considered as atypical in terms of the anticipated level of control resulting from a good protective spray schedule. Relative differences between treatments, however, are probably applicable in any schedule.

Powdery mildew control was evaluated 15 times at 1-week intervals beginning with a pretreatment evaluation on April 12.

The amount of mildew present was determined from a total of 100 terminals per tree, selected at random primarily from the periphery of the tree at shoulder height as the tree was completely circled. Some terminal selections were made "inside" the tree. Infection density was so high throughout the entire season that no observable differences in infection existed with respect to location of terminals. A "terminal" was at least 4 inches in length and bore an average of four leaves. The terminal was rated as infected if active mildew was visible on any leaf. Infection counts were reported in terms of percentages.

DISCUSSION OF RESULTS

Virginia

A preliminary mildew count was made on May 20 at which time three sprays at weekly intervals, and two each at the 2- and 3- week intervals, had been applied. Five leaves per station were scored at this time in accordance with the method used in 1956. Highly significant inter-treatment differences were found to be inherent in the data. The pattern of infection was such that the replicates differed substantially in disease level. These differences in disease levels between replicates were removed from inter-treatment comparisons.

Spray interval was more important than dosage in both the preliminary and the final Virginia mildew counts. Both dosage and spray interval are significant in both the preliminary and final counts. Over the range of the four dosages employed, the effects of different spray intervals were demonstrable in the averages; lengthening the time interval between sprays invariably resulted in increased lesion counts. Over the range of spray intervals employed, the effect of rate of usage was also clearly apparent; reducing the amount used invariably resulted in increased lesion counts.

A final and more extensive mildew count was made on July 1 and 2. Nine sprays had been applied at weekly intervals, five sprays at 2-week intervals and three sprays at 3-week intervals. The count followed the final spray by 1 week. A summary of the final counts is given in Table 1.

The final data confirmed the preliminary findings. Differences between treatments were demonstrable at the 1 percent confidence level. Valid differences in infection levels between replicates were still present, although the progress of infection was such that the magnitude of replicate differences had decreased substantially from that of the preliminary count. Infection within a tree was still uniform.

When sprays were applied at weekly intervals the 1-pound dosage resulted in near perfect mildew control (99.7 percent). The 12- and 8-ounce rates also gave excellent control. Although the 4-ounce rate gave statistically significantly poorer control (94.5 percent), the practical importance of the difference is questionable. The degree of control afforded by even the lowest rate of usage at weekly intervals under the conditions of this experiment is considered to be commercially acceptable.

When sprays were applied every 2 weeks, control was still good although there was a progressively greater number of mildew infections as the rate of usage declined. The control afforded by the 1-pound rate at the 2-week interval was approximately the same as that which

resulted from the 4-ounce rate applied weekly. Similarly, the 4-ounce rate applied every 2 weeks gave as good control as did the 1-pound rate applied every 3 weeks.

When sprays were applied every 3 weeks, a further loss in mildew control resulted. This loss may not be valid except at the lower rates of usage.

Spray interval has a substantial effect on the performance of Karathane over the range of dosages employed. A twelvefold difference in the lesion count between interval extremes is demonstrable. Dosage also has an effect over the range of spray intervals employed, but only a threefold difference in lesion counts is evident.

The effect of the time interval between sprays emerges as the most important factor in determining the biological effectiveness of Karathane in these trials. Although both rate of usage and spray interval are still considered to be important, the interaction of rate-of-usage X time-interval is no longer significant, and thus the relative effect of dosage was the same (within the limits of error) with all spray intervals at the conclusion of this experiment.

Both the Virginia and California data were examined in graphic expression. The inflection point of the average dosage response curve seemed to lie between the 8- and 12-ounce rates of usage in the Virginia data. In the individual curves the inflection point lay between 4 and 8 ounces for the weekly spray interval, and between 8 and 12 ounces for the two longer time intervals. This suggests that the inflection point moves, depending upon the time interval between sprays. The inflection point of the dosage response curves in the California 2 (Barnes Manzanita) experiment seemed to lie consistently at the 8-ounce rate in both the individual and average curves. This makes it difficult, if not impossible, to select a single optimum dosage recommendation applicable to all circumstances.

Foliar residue analyses accompanied the 1956 Virginia trials to develop information on the deposition and degradation rates for Karathane. The initial deposit was found to be directly related to the concentration applied, and the rate of degradation to be rapid. The half-life of Karathane was between 1 and 1.5 days. For all practical purposes, residues were negligible after 4 days. Long lasting or persistent residues either do not occur, or are in a form not detectable by the analytical method used. Heavy deposits apparently cannot be accumulated through repeated applications at the time intervals employed.

California

A summary of the mildew evaluation data from the Barnes Manzanita Ranch is given in Table 2.

When Karathane applications were made at weekly intervals in this experiment there was no difference in mildew control of practical importance between the 8-, 12-, and 16-ounce rates of usage. The 4-ounce rate was significantly inferior to the three higher rates of usage at the 5 percent confidence level, although it gave almost 95 percent control of the disease.

When the sprays were applied at 2-week intervals, the influence of rate of usage became more obvious; the higher the dosage, the better the disease control. There is the suggestion of a sharp break in disease control effectiveness between the 4- and 8-ounce rates of usage for both the 1- and 2-week time intervals.

When treatments were applied every 3 weeks, all concentrations of Karathane gave unsatisfactory mildew control. A differing dosage response had disappeared except for the 4-ounce rate which was significantly less effective than the three higher concentrations.

The influence of spray interval over the range of dosages employed is obvious in the averages. There is an elevenfold increase in the disease rating when the time intervals are increased from 1 to 3 weeks. Over the range of spray intervals employed, the effect of rate of usage is also obvious. The magnitude of the dosage effect is considerably less than that of spray interval, however. A two-and-one-half times increase in disease rating resulted when dosage was reduced from 16 ounces to 4 ounces. The relative importance of the spray timing interval as compared with that of rate of usage was demonstrable in each of the weekly evaluations.

The conclusion to be drawn from this experiment appears to be that the most efficient way to utilize Karathane for powdery mildew control is to apply small amounts at frequent intervals. The results as revealed in these data suggest that the optimum treatment from a practical standpoint to be about 8 ounces applied every 2 weeks. The possible increased eradicative effectiveness of higher dosages might be justified where it is necessary to cope with an established severe infection.

The experiment at the W. L. Farmer Ranch was the most rigorous test of the series reported. The orchard in which the test blocks were located had a history of severe infection,

Table 1. The average effect of Karathane applied at three time intervals and four rates of usage on the incidence and control of apple powdery mildew; O'Brien Orchard.

Oz per 100 Gal.	7-day intervals			14-day intervals			21-day intervals			Averages		
	Rating	Log*	Control	Rating	Log*	Control	Rating	Log*	Control	Rating	Log*	Control
4 oz.	0.14	1.30	94.5%	0.35	1.74	86.5%	1.08	2.23	58.4%	0.53	1.76	79.8%
8 oz.	0.04	0.83	98.4%	0.34	1.71	87.0%	0.64	2.01	75.0%	0.34	1.51	86.8%
12 oz.	0.03	0.50	99.0%	0.17	1.37	93.5%	0.35	1.63	86.7%	0.18	1.17	93.1%
16 oz.	0.01	0.25	99.7%	0.11	1.24	95.9%	0.38	1.50	85.4%	0.16	1.00	93.7%
Average	0.05	0.72	97.9%	0.24	1.52	90.5%	0.61	1.84	76.4%	0.30	1.36	88.3%

Captan standard schedule without a mildew fungicide	2.60	00.0%
Captan standard, sprayed with water only every week	2.40	7.8%
Captan standard, sprayed with water + B-1956 every week	1.99	22.8%

LSD (.05) Between Individual Treatments..... 0.43

LSD (.05) Between time Interval Averages..... 0.22

LSD (.05) Between Dosage Averages..... 0.25

* Log values refer to the average logarithm of the total number of lesions per replicate plus 1. These values should be used in estimating the significance of inter-treatment comparisions.

Note: The rating values listed here are the mean number of discrete mildew lesions per leaf.

Table 2. The average effect of Karathane applied at three time intervals and four rates of usage on the incidence and control of apple powdery mildew; Barnes Manzanita Ranch.

Oz. per 100 Gal.	7-day intervals			14-day intervals			21-day intervals			Averages		
	Rating	Log*	Control	Rating	Log*	Control	Rating	Log*	Control	Rating	Log*	Control
4 oz.	0.10	1.31	91%	0.34	1.85	69%	0.45	1.95	58%	0.30	1.70	72%
8 oz.	0.03	0.78	97%	0.15	1.47	86%	0.29	1.74	73%	0.16	1.33	85%
12 oz.	0.02	0.61	98%	0.08	1.19	93%	0.32	1.62	70%	0.14	1.14	87%
16 oz.	0.01	0.23	99%	0.04	0.86	96%	0.24	1.66	78%	0.09	0.92	92%
Average	0.04	0.73	96%	0.15	1.34	86%	0.32	1.74	70%			
							Untreated Control.....			1.08.....		0.0%
LSD (.05) Individual Treatments					0.30							
LSD (.05) Between Week Intervals Average					0.15							
LSD (.05) Between Dosage Averages					0.18							

* Log values refer to the average logarithm of the sum of observations from the 7 evaluations. These values are not the logs of the ratings indicated but should be used in estimating the significance of inter-treatment comparisons.

Table 3. The average effect of Karathane applied at three time intervals and four rates of usage on the incidence and control of apple powdery mildew; W. L. Farmer Ranch.

Oz. per 100 Gal.	7-day intervals		14-day intervals		21-day intervals		Averages	
	Rating	Control	Rating	Control	Rating	Control	Rating	Control
4 oz.	47.8	42.7%	70.6	15.4%	70.2	15.9%	62.9	24.7%
8 oz.	36.4	56.4%	64.0	23.3%	68.8	17.6%	56.4	32.5%
12 oz.	31.4	62.4%	54.1	35.2%	67.9	19.7%	51.1	38.8%
16 oz.	25.4	69.6%	52.5	37.1%	64.7	22.5%	47.5	43.1%
Average	35.3	57.7%	60.3	27.8%	67.9	18.7%		
						Untreated Control.....	83.5.....	0.0%
LSD (.05)	Between Individual Treatments			6.0				
LSD (.05)	Between Week Interval Averages			3.0				
LSD (.05)	Between Dosage Averages			3.5				

Note: The rating values listed here are the mean number of infected terminals per 100 counted.

Table 4. Phytocidal effects of Karathane to apple foliage when applied at four concentrations during average daily temperature maxima of 94° F; O'Brien Orchard, Berryville, Virginia.

Rate of Usage per 100 Gallons	Average Injury Rating*
4 oz.	0.01
8 oz.	0.04
12 oz.	0.18
16 oz.	0.36
Control, no Karathane	0.00
LSD (.05) Between Treatments	0.099

Note: Each value represents the mean of 640 observations, the same as for the infection counts.

* Rating System: 0 = No injury

1 = No injury on upper leaf surface, trace of discoloration on lower surface.

2 = Trace of discoloration on upper surface, 10 to 50% discoloration on lower surface. No higher degree of injury was present.

and an average of 62.2 percent of the terminals were determined to be infected at the time this first test spray treatment was applied. A total of 14 weekly sprays were applied. The status of mildew infection was evaluated 15 times. A summary of the several evaluations is given in Table 3.

The influence of the time interval between sprays, regardless of fungicide concentration, is quite striking in this experiment. Where sprays were applied at weekly intervals an average of 35.3 percent of the terminals became infected. Where the time interval was increased to 2 weeks, an average of 60.3 percent of the terminals became infected. The average level of infection increased slightly, to 67.9 percent, at the 3-week interval. A treatment of 4 ounces applied weekly was superior to any rate of usage at a longer time interval.

The influence of rate of usage is most apparent at the weekly time interval. There is a slightly less than twofold difference between the 4- and 16-ounce dosage at weekly intervals. At 2-week intervals the differences are still present, but reduced. Differences between the rates of usage at the 3-week interval are small and of questionable significance.

The results of this experiment also tend to indicate that the most efficient way to utilize Karathane is to apply small amounts at frequent intervals. None of the treatments was considered to be satisfactory for the control of mildew under the conditions of this experiment and as judged by the evaluation method used. Failure to classify degrees of infection may have obscured real differences. The method necessarily minimizes differences and weights the count in favor of the poorest treatment.

Karathane might have performed more creditably in this experiment if spray applications had been strated earlier in the season, thereby reducing infection on newly emerging bud and leaf tissue and thus avoiding the extremely high infection levels encountered at the full pink stage, when the first spray applications were made. Considering the serious mildew infection history of the orchard in which this experiment was conducted, a comparison of a delayed dormant with a pre-pink application would have been of interest in respect to their effect upon subsequent infection levels.

The best showing for all treatments was made after four weekly applications when the average level of infection had been reduced from an initial average level of 63.4 percent to 30.8 percent for all dosages. The reduction for the 1-pound rate was from 67.7 percent to 11.5 percent at this time. The infection level fluctuated for the remainder of the season. Aside from any consideration of the fungicidal capabilities of Karathane, other factors which are considered to have influenced the results of this experiment are the inability to provide area protection from adjacent severly infected trees, weather conditions which prevent adherence to all planned time intervals, and the late start of the trials.

CONCLUSIONS

The following general conclusions regarding the effective use of Karathane for control of apple powdery mildew are offered on the basis of experience gained in and under the conditions of these experiments.

1. Eight ounces of formulated Karathane per 100 gallons of spray is adequate for mildew control with proper timing of sprays.
2. The time interval between sprays should not exceed 2 weeks. More frequent applications may be required early in the season or to obtain satisfactory control under severe disease conditions.
3. Shortening the time interval between sprays is preferable to increasing the rate of usage as a modification to bring severe disease conditions under control.
4. As little as 4 ounces of Karathane may prove adequate for mildew control under some conditions or on less susceptible varieties, provided the time interval is not over 1 week.
5. The phytotoxic potential of Karathane can be minimized through the use of low dosages (8 ounces or less) and avoiding application at temperatures above 90° F. Maximum temperatures for 36 hours following an application is of major importance in producing injury.
6. The normal protective spray schedule recommended for scab and rust protection in the East provides a satisfactory time interval for mildew control where Karathane is added. Applications should begin in the pre-blossom period.

VIRGINIA AGRICULTURAL EXPERIMENT STATION; AND RESEARCH DIVISION, ROHM AND HAAS COMPANY

GROWTH OF PEACH TREES IN SOILS TREATED ELEVEN YEARS PREVIOUSLY WITH DDT

John C. Dunegan and Roy A. Wilson

In January 1946, M. C. Goldsworthy mixed technical DDT[1], in amounts varying from 0 to 1600 pounds of the chemical per acre, into soil of three types at Plant Industry Station, Beltsville, Maryland. This DDT-treated soil was used in a series of greenhouse experiments and in 1948 Goldsworthy (1) reported that the presence of DDT in the soil affected the growth of 1-year-old Elberta peach trees.

> "....This effect varied somewhat with soil type. In the heavy Chester soil, reduction of growth was statistically significant at the 200-pound level; in the Sassafras soil, at the 800-pound level; and in the Evesboro soil significant reduction was evident for the 25-pound application, and again with the 200-pound application. At the 50- 100-pound levels of application in all three soils the growth of roots and tops was practically as good as or better than that in untreated soils. In all cases where less top growth occurred less root growth was also observed...."

The various soil samples were subsequently stored in a greenhouse basement, at the Plant Industry Station, and left dry and undisturbed from 1948 to 1957.

On March 4, 1957 the soil samples were removed from storage. All the replicates of each treatment were thoroughly mixed together, and a new randomized experiment involving five replicates of each treatment of the three soils was set up in the greenhouse. The soils were moistened and dormant 1-year-old Lovell seedlings were planted, one per pot, on March 11. Each seedling was weighed just before it was planted and again on June 30, at the end of the experiment. All leaves were removed and the soil washed from the roots prior to the final weighing. The weight of each group of replicates is given in Table 1.

No symptoms of retarded growth, such as observed by Goldsworthy in the earlier studies, were noted during the course of the experiment. Likewise the root systems of the trees from the treated soils did not differ in appearance from those of trees grown in the non-treated soils.

However, there was some variation in the weight increment among the various replicates at a given DDT level as well as among the different levels on each soil type. These data were studied statistically by E. J. Koch, of the Biometrical Services, Plant Industry Station. He found there was a significant variation in the replications with a constant trend from replicate one to five in the various groups. The cause for this variation is obscure, but may be the result of some position gradient, even though the different replicates were arranged at random on the greenhouse bench. The soil types were found, as was expected, to be significantly different. Growth on the Evesboro loamy sand was better than on the Chester heavy clay loam. There was no evidence, however, of any significant difference between the various amounts of DDT added to the soil in relation to the amounts of tree growth except that all but one of the DDT treatments caused a slight stimulation in growth. However, the amount of stimulation was not correlated with the amount of DDT applied. Further studies (covariance analysis and the calculation of correlation coefficient) showed there was little relation between the original weight of the trees and the final weight. The variations noted at the termination of the experiment were therefore not a reflection of the differences in the weight of the trees as they were planted.

All the evidence available from this experiment indicates that although certain amounts of DDT added to soils remained toxic for at least two years, this effect had dissipated by the end of 11 years under the conditions of this experiment (i.e., soil stored dry and undisturbed for 9 years).

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[1] The material used was a commercial formulation called Deenate 25W containing 75% clay and 25% technical dichloro diphenyl trichloroethane.

Table 1. Growth of Lovell peach seedlings in soil treated 11 years previously with various amounts of DDT.

Soil type and amount of DDT added (pounds per acre)	Total weight of 5 plants (grams)		Gain in weight (grams)
	March 11, 1957	July 30, 1957	
Chester heavy clay loam			
0	41	633	592
25	56	1169	1113
50	51	538	487
100	56	825	769
200	63	815	752
400	55	861	806
800	56	744	688
1600	50	743	693
Sassafras sandy loam			
0	67	573	506
25	56	859	803
50	82	1069	987
100	56	991	935
200	49[a]	731	682
400	62	994	932
800	49	849	800
1600	61	929	868
Evesboro loamy sand			
0	49	907	858
25	58[a]	1224	1166
50	45	1037	992
100	64	1169	1105
200	58	1441	1383
400	59	1231	1172
800	57	943	886
1600	56	1170	1114

[a] One tree failed to grow. Weights are based, therefore, on four instead of five replicates.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

THE OCCURRENCE OF OÖSPORA ROT OF CITRUS IN LOUISIANA[1]

Norman L. Horn, J. B. Sinclair, and R. T. Brown[2]

A fruit rot was found in Louisiana citrus groves during the late summer and fall of 1957. The condition followed an extended rainy period and was observed first on navel oranges. These observations were made in September, when navel oranges are normally totally green. Because the diseased fruits tended to ripen prematurely, infected oranges were easily identified on the trees. Upon further survey, both satsumas and kumquats were found to be affected (Fig. 1). Infected fruit were observed not only on the ground and lower limbs of the trees but also on the highest branches.

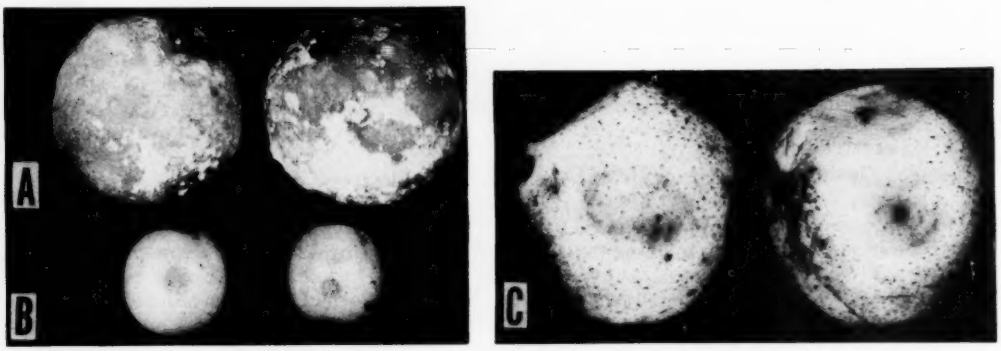

FIGURE 1. Oöspora rot of citrus. A -- Natural late infection on satsumas. B -- Natural early infection on kumquat. C -- Artificially infected navel oranges.

Eight growers were consulted concerning the estimated losses resulting from the disease under observation. Reported losses ranged from 10 to 30 percent of the total crop. At the Plaquemines Parish Experiment Station, Port Sulfur, Louisiana, damage was approximately 20 percent.

A fungus belonging to the genus Oöspora was consistently isolated from infected fruit of navel orange, satsuma and kumquat. Green navel oranges were inoculated with isolates of the organism by puncturing the fruit with a sterile needle that had been dipped in a conidial suspension of the fungus (Fig. 1 C). The fruit were then placed in a high humidity chamber maintained at room temperature. Within 3 days spots approximately 1 inch in diameter appeared at each puncture with a white mycelial growth developing from the points of infection. Most of the oranges had developed typical soft rot 7 days after inoculation (Fig. 1 C). They were sour but not too pungent. The rot was not extremely watery, so that the fruit remained intact when handled. Fruit punctured in the same manner, but without the conidial suspension, did not show comparable rotting at any time. These results do not agree with the report of Smith[3], who indicated that Oöspora citri-aurantii, which incites a storage rot of lemon, was a putrid, sour, leaking, soft rot. He reported also that inoculated fresh green fruit did not become infected readily. The characteristic rot was not produced when a conidial suspension was placed on green navel oranges without puncturing the fruit.

[1] The authors wish to express their appreciation to Dr. M. P. Bachus, Department of Botany, University of Wisconsin, for confirming the identification of the causal organism, Oöspora sp.
[2] Associate Plant Pathologist, Assistant Plant Pathologist, and Superintendent, Plaquemines Parish Experiment Station, respectively, Louisiana State University Agricultural Experiment Station.
[3] Smith, C. O. 1917. Sour rot of lemon in California. Phytopathology 7: 37-41.

The morphological characters of the organism isolated in Louisiana, when cultured on green navel oranges, resembled closely those described by Smith[3]. On potato-dextrose agar the fungus was morhplogically different. In the latter case conidia measured 3.3 x 3.3 μ to 3.9 x 14.9 μ. Cells were spherical, oval and oblong with some of the latter two types having obtuse ends. All conidia examined contained from two to many oil globules. The cultures on potato-dextrose agar were off-white, much depressed and restricted to the surface of the medium. The sparse mycelium produced an abundance of conidia appearing as powdery masses on the edge of the colonies.

As far as could be determined, this is the first report of Oöspora sp. causing a fruit rot of citrus in the field in Louisiana.

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MANEB-OIL INJURY TO GRAPEFRUIT[1]

Fran. E. Fisher

The spectacular experimental control of citrus fruit russet and greasy spot with a single summer application of zineb, in 1956[2], and the still more spectacular commercial control with the same material, in 1957[3], has led to considerable speculation regarding the use of other fungicidal materials, especially the carbamates, for controlling these serious maladies. In particular, the writer has heard of several instances in which maneb has been recommended for commercial use in preventing russet. Although maneb was one of the materials found to decrease the incidence of russet in the experimental trials of 1954[2], there are good reasons why it should not yet be so recommended or used. These are: 1) no tolerance for maneb on citrus has been established by the U. S. Food and Drug Administration, 2) maneb is more costly to use than zineb, and 3) better and less expensive control of greasy spot can be obtained with zineb, copper, or oil emulsion[4]. In addition, it has now been found that maneb used in combination with oil causes severe injury to fruit of grapefruit and somewhat less severe injury to fruit of Valencia orange. It is usually necessary to apply oil or some other insecticide during the summer months for the control of scale insects. In the commercial production of citrus it is advantageous to use a fungicide compatible with oil so as to economize on cost of application. This paper describes the experiments in which oil-maneb sprays resulted in severe injury to grapefruit.

In the summer of 1957, maneb-oil sprays were applied to groves of the following varieties located over a 50-mile area of central Florida: Duncan, Silver Cluster, and Ruby Red grapefruit and Valencia orange. In a typical experiment, a grove of Duncan grapefruit trees was divided into plots of four trees each, randomized, and replicated four times. Tests on a commercial basis consisted of alternating the types of materials on plots made up of five rows of 20 trees. All trees received a single application between June 24 and July 12.

Beginning September 17, all plots were examined. Injury to grapefruits was found to occur in all trees treated with maneb-oil (Fig. 1). This injury was not found to occur in trees that received oil-emulsion or zineb-oil sprays, nor in non-sprayed trees. Injury was very slight and difficult to detect on maneb-oil-sprayed Valencia oranges.

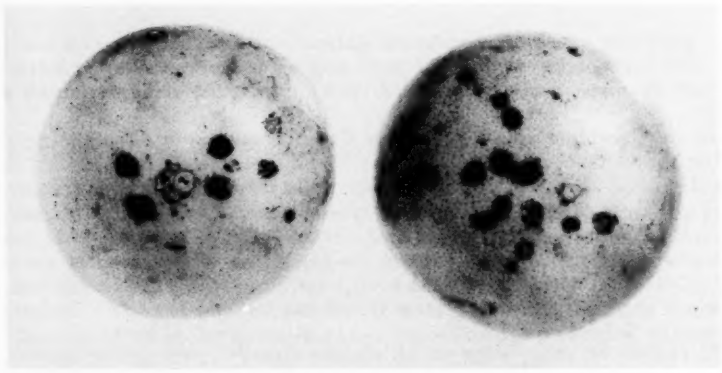

FIGURE 1. Injury on the blossom-end of Ruby Red grapefruits resulting from the use of 1 pound maneb-1.3 percent oil.

[1]Florida Agricultural Experiment Station Journal Series, No. 716.

[2]Fisher, F. E. 1947. Control of citrus fruit russet in Florida with zineb. Phytopathology 47: 433-437.

[3]Miller, R. L. 1947. Zineb-improved summer control of citrus fruit russet. Citrus Industry 38: 7-9, 27, 28.

[4]Fisher, F. E. 1955. Greasy spot. Florida Agricultural Experiment Station Annual Report p. 184.

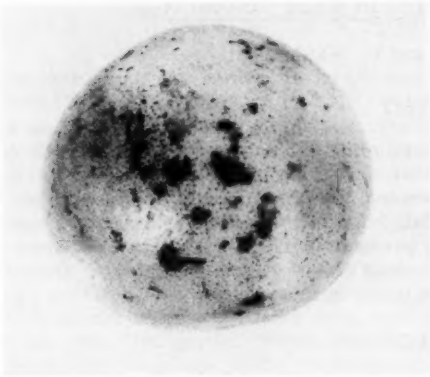

FIGURE 2. Injury on exposed side of Silver Cluster grapefruit following the use of 1 pound maneb-0.7 percent oil.

Injury following maneb-oil sprays was confined primarily to the lower or exposed portions of the fruits. The blemish was superficial on the rind and ranged in color from tan to very dark brown. No appreciable injury to leaves was detected. However, scar tissues present on leaves at time of spraying were made more prominent, as is also the case whenever copper-containing materials are used. The combination of 1.3 percent oil and 1 pound maneb resulted in more severe injury (Fig. 1) than did 0.7 percent oil plus 1 pound maneb (Fig. 2).

Although the summer application of maneb-oil results in injury to grapefruit, maneb applied alone or in combination with materials other than oil eventually may be practicable. At this time, however, maneb can not be recommended for use on citrus.

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AN ATTEMPT TO EVALUATE THE SIGNIFICANCE OF DEAD OAK TREES FOUND IN OAK WILT SITES IN WEST VIRGINIA[1]

William H. Gillespie and F. Waldo Craig[2]

Summary

The number of dead oaks occurring in 1572 naturally infected oak wilt sites is contrasted with those occurring in an equal number of randomized, apparently non-diseased, plots. The data are interpreted to mean that, in West Virginia, the finding of two or more dead trees near an actively wilting oak tree or trees will, in a majority of cases, indicate that the dead trees were killed by oak wilt (Ceratocystis fagacearum) sometime in the past.

INTRODUCTION

Since 1944, when the first article on the significance of oak wilt (Ceratocystis fagacearum (Bretz) Hunt) was published (8), many descriptions have been written of wilting oak trees surrounding pockets of dead trees. The accepted interpretation has been that such trees are killed radially from a new center of infection and that, after several years, a pocket of dead trees is formed, surrounded by wilting trees (5). This assumption is more or less borne out by a 10-year study conducted in Wisconsin, where the disease followed such a pattern of spread in 38 of 41 plots (9). Numerous reports from agencies within States where oak wilt control is attempted give additional support to this view.

In one publication (11) it is inferred that groups of dead oak trees, lacking associated actively wilting trees but occurring in areas containing oak wilt at other points, should be regarded as having died of the disease, which had since ceased to spread locally. Another paper (10) requests that all such clumps of dead trees be reported to the control agencies, unless obviously killed by fire. In the same paper, a single dead tree is not considered important.

The presence or absence of dead oak trees near oak wilt sites has been used as evidence in interpreting the history of the disease in particular localities. For example, surveys conducted in some areas to determine the amount of damage caused by oak wilt have utilized dead trees as partial evidence of the occurrence of the disease (1). In another instance dead trees were used as a criterion for differentiating between areas which showed differences in root grafting potential (6). Finally, it has been suggested that the age of an individual infection can be determined by ascertaining the time of release within the site of trees and shrubs which were previously suppressed by the killed oaks (4). The validity of each of these determinations lies in the supposition that the dead trees within the site were killed by oak wilt.

Few observations have been reported in regard to whether such dead trees, in sites which have not been under specific observation, are actually old oak wilt trees. Bretz (2) says that "the bark of wilt-killed trees loosens rather rapidly and, by the end of the second year following death, may begin to shed from the bole." This characteristic was also described by Brinkman (3) who reported that northern red oak trees, dying from oak wilt in an unmanaged Iowa forest, lost their bark early and remained standing for 7 or 8 years. This same early shedding of bark has been observed in West Virginia on dead trees whose death was attributed to oak mortality (7). Therefore, the loss of bark, although often associated with oak wilt, is considered to be of secondary importance in diagnosing the cause of death of a particular tree.

When an attempt was made in West Virginia to determine how many of the dead trees in oak wilt sites actually died as a result of the disease, no method could be found which would prove they had been killed by wilt. Loose bark gave only an indication, mat scars were seldom present and even less reliable, and increment cores showed no pattern that could be consistently associated with the death of the trees. Since more than one-half of the new sites found in recent years in West Virginia have contained one or more dead trees, the following procedure[3] was selected as a step toward the development of a method that would aid a control pro-

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[1]Published with the approval of the West Virginia Commissioner of Agriculture as Special Survey Paper No. 4.

[2]Oak Wilt Coordinator, and Chief, Plant Pest Control Division, West Virginia Department of Agriculture, respectively.

[3]Preliminary results were noted in the 1956 report of the West Virginia oak wilt program.

gram in evaluating the significance of these dead trees.

METHODS

An intensive oak wilt control program was carried out in West Virginia during the summers of 1956 and 1957. This program made it possible to visit a majority of the known oak wilt locations and to find 1572 new sites[4]. An analysis of the data collected at each of these new locations showed that 51.3 percent of these sites contained at least one dead tree in addition to the currently wilting trees.

As one method for investigating the importance of this figure, it was decided to study an equal number of randomized, non-diseased plots and to compare the data from the two groups. Various counties were selected for this work, the only criterion being that they must be located in sections containing oak wilt so that the sectional environment would be as nearly comparable as possible. An observer then drove to previously selected regions within these counties and, from arbitrarily chosen points, walked into the woods a certain distance (i. e. the thirtieth oak tree, 100 yards, 300 yards, etc.) which was also decided in advance, and found the nearest healthy oak. All dead trees within an estimated 50 feet of this oak were then tallied.

RESULTS AND DISCUSSION

The data from the 1956 and 1957 oak wilt programs are compared in Table 1, both separately and together, with data from the randomly chosen non-diseased sites.

These data show striking differences. For example, 48.7 percent of the oak wilt sites, when first found, contained only a single wilting tree with no associated dead oaks. This compares with the 94.3 percent of the random controls which had no dead trees. Similarly, 51.3 percent of the wilt sites included dead oak trees as compared with only 5.7 percent of the check plots. The percentages seem even more significant when the sites are categorized into those with only one dead tree and those with two or more. Of the oak wilt plots 21.5 percent had only one dead tree associated with the actively wilting tree while 29.8 percent had two or more dead trees. This contrasts with the 4.5 percent of the check plots which contained only one dead tree and the 1.2 percent which contained two or more.

Many of the dead trees found at the non-diseased plots could be attributed to lightning, earthslips, exposed roots, etc. This was not true of most of the dead trees associated with the oak wilt sites.

If these data are considered as relevant evidence, then, in West Virginia, the finding of one dead oak tree associated with an actively wilting tree will indicate, in three out of four instances, that the dead tree was probably killed in previous years by the disease. Similarly, the finding of two or more dead trees near an actively wilting tree will, in about 29 out of 30 times, indicate that the dead trees were previous victims of oak wilt. The mean number of dead trees per oak wilt site in this study was 1.1 as contrasted with 0.07 for the non-diseased plots. The maximum number of dead trees found in an oak wilt site was 53.

These specific figures may have no significance in other States, especially in areas where the oaks are on marginal sites or are attacked by other diseases, but they will be helpful in answering certain questions that have arisen in West Virginia. For example, several localized areas containing groups of five, six, seven, or more, dead trees which showed no external cause for their death were plotted during the 1955 season. These areas were observed several times during succeeding oak wilt surveys, and in 1957 actively wilting trees were found around some of them. Perhaps smaller groups, containing only two or three dead trees which show no evident reason for their death, should also be mapped and observed annually.

The above interpretations are subject to change and qualification as new facts become available. Oak mortality has killed hundreds of oak trees in parts of West Virginia over the last two or three years. These data would not be applicable in such areas, but the method is presented as another way of gathering evidence for the evaluation of certain oak wilt control results.

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[4]A site is a circle with a 50-foot radius around an oak wilt tree or around a group of intermingled dead and wilting oaks each of which is within 50 feet of another.

Table 1. Comparison of data on occurrence of dead oak trees in oak wilt sites and in randomized non-diseased check plots, West Virginia.

Source of data	Total number of new sites	Single tree sites		Dead tree sites					
				Total		With one dead tree		With two or more dead trees	
		Number	Percent	Number	Percent	Number	Percent	Number	Percent
Oak Wilt Sites									
1956 oak wilt survey	890	431	48.4	459	51.5	186	21	273	30.5
1957 oak wilt survey	682	334	49.0	348	51.0	152	22	196	29
Combined 1956-57 data	1572	765	48.7	807	51.3	338	21.5	469	29.8
Randomized Check Plots	1572	1483	94.3	89	5.7	71	4.5	18	1.2

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PLANT PEST CONTROL DIVISION, WEST VIRGINIA DEPARTMENT OF AGRICULTURE

A NEW LEAF SPOT DISEASE OF MISTLETOE IN FLORIDA

Harry C. Burnett[1]

On December 18, 1957 a clump of mistletoe, Phoradendron flavescens (Pursh) Nutt., was observed on the campus of the University of Florida, Gainesville, Florida, infected with a leaf spot. Further examination revealed that a species of Cercospora was fruiting on these spots. Material was sent to Dr. Charles Chupp, who said the spots are identical with spots caused by Cercospora struthanthi Chupp & Muller, found in Venezuela on Struthanthus sp. in 1941, and the organism is so very similar that he believes it is the same species.

Chupp, in his "A Monograph of Cercospora", published in 1953, described the spots on Struthanthus sp. as circular, 5-20 mm in diameter, grayish-brown to almost black, bordered by a narrow, pale brown raised line.

A quick survey of Phoradendron flavescens growing within a radius of 8 miles of Gainesville was made. In all, over 25 clumps of this mistletoe growing on species of Quercus and Prunus were noted, and each one was found infected with Cercospora struthanthi. This leafspot causes considerable defoliation. A diagnosis of a diseased plant may be made by the fallen leaves showing infection.

Infected leaves have been deposited in the Florida Agricultural Experiment Station Herbarium under # F-46422.

Figure 1 shows symptoms of the disease on Phoradendron flavescens.

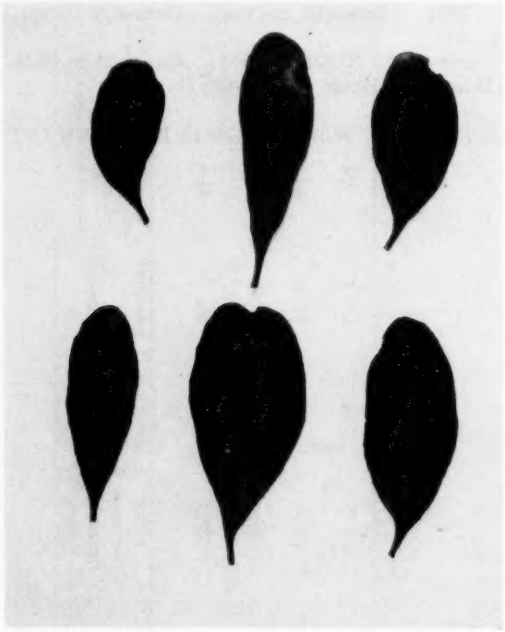

FIGURE 1. Symptoms of Cercospora struthanthi Chupp & Muller on mistletoe, Phoradendron flavescens (Pursh) Nutt. (State Plant Board Photo by Jean Smith).

STATE PLANT BOARD OF FLORIDA, GAINESVILLE, FLORIDA

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SOIL FUMIGANTS AND THEIR USE: A SUMMARY[1]

G. K. Parris[2]

INTRODUCTION

The accumulation of various pests in the soil, namely fungi, bacteria, nematodes, insects, and weeds has reduced the productive capacity of vast areas of otherwise good land. Soil can be rid of these pests by using volatile chemicals some of which are relatively inexpensive. Modern methods and machinery have increased the ease of application and reduced the danger of handling these soil fumigants, and lowered costs. As the cost of soil fumigation has been lowered the areas of usefulness have enlarged. The process of soil fumigation technically is "soil disinfestation," but it is commonly called "soil disinfection."

THE NATURE AND ACTION OF SOIL FUMIGANTS

A soil fumigant is an organic chemical, either a liquid of high vapor pressure which may or may not have a low boiling point necessitating it be kept under pressure when not in use (e. g. methyl bromide), or a liquid absorbed on some inert powder such as bentonite clay or talc which acts as a carrier. The combination of liquid fumigant plus carrier changes the powder to "granules." Fumigant liquids, if exposed, soon evaporate. The "granules" lose their potency unless kept in tightly closed containers. The speed of evaporation or volatilization is determined by the character of the chemical and the temperature. In practice, a fumigant is introduced into holes in the soil, usually at a depth of 6 inches and at regular intervals 12 to 18 inches apart, or it may be trickled or dripped into furrows, 6 inches deep and 12 to 18 inches apart, by special application machinery. This can be done by hand on small scale operations. If the fumigant is absorbed on an inert carrier the granules may be broadcast and then plowed under (4 to 6 inches deep), or applied beneath the soil in bands, 4 to 6 inches deep and 12 to 18 inches apart, by fertilizer or seed drills. The holes or furrows into which the fumigant is introduced should be closed immediately with adjacent soil, and the treated soil either left uncovered, or covered with a seal that is gas-tight. Whether or not a seal is needed depends on the volatility of the soil fumigant. Chloropicrin and methyl bromide require a surface seal, while D-D mixture, ethylene dibromide, "Nemagon", "Vapam", "Terraclor", "VC-13 Nemacide", "Mylone", and "Telone" require no seal. The seal may be asphalt (tar) mulching paper which is expensive but very effective, or a thorough soaking of the upper 2 inches of the treated soil with water (one to 2 quarts per square foot). Glue-coated paper is a cheap substitute for asphalt paper. The volatile chemical diffuses through the soil in all directions, but most vigorously upwards, and destroys soil-borne pests in its path.

The soil pests are killed presumably by action of the fumigant on the protoplasm of the cells. Chloropicrin destroys all types of protoplasm, plant as well as animal; D-D mixture, ethylene dibromide, Nemagon, VC-13 Nemacide, and Telone, act only on animal protoplasm, specifically that of nematodes; methyl bromide shows promise against nematodes, weed seeds, and many fungi; while Vapam and Mylone are said to control nematodes, certain fungi, and weeds; Terraclor acts only as a fungicide.

TYPES OF SOIL FUMIGANTS: THEIR VALUES AND THEIR LIMITATIONS

A number of chemicals have been tried in the past 20 years as soil fumigants. Many of these have been discarded for good reasons and will not be mentioned. Those that have stood the test of time, convenience, and economy, as well as the newest ones, will be compared here. Ahead lies the perfect soil fumigant, -- one that is cheap ($20 to $30 per acre including cost of application), is easy and safe to apply, is retained fairly readily by soil without the necessity of a seal, is not injurious to the tops or roots of living plants in or near the treated soil, and is effective at temperatures of 40° to 70° F against fungi, bacteria, nematodes, and weed seeds. This development is not outside the ability of man to devise. Within the past ten years, research by commercial companies and also by experiment station workers has developed soil fumigants that can be safely applied to soil in which living plants

[1]January 2, 1952; revised December 10, 1957.
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are established. The fumigant gets rid of the noxious pest without measurable plant toxicity. Such materials are especially useful in the control of pests attacking valuable perennial plants. Fumigants of this character often show specific, differential effects on plants.

CHLOROPICRIN: This liquid, trinitro methane, commonly known as "tear-gas" is also called "Larvacide" which is a trade name (Innis Speiden Co., New York City). It is used at rates of 300 to 400 pounds per acre. The high cost per acre ($400) limits the use of chloropicrin; however, it is extremely effective against fungi, nematodes, insects, and weed seeds. It is not as effective against unrotted nematode galls as desired. A surface seal of treated soil is necessary; either paper or water may be used (see above). Chloropicrin will injure or kill aerial as well as subterranean parts of any plants present in the area of application and cannot be used in a greenhouse and should not be used near established plants out-of-doors. Chloropicrin is corrosive to machinery and extremely unpleasant to handle and apply. However, with applicators and proper instructions as to handling, chloropicrin can be used with no danger to the operator. It should not be applied unless the soil temperature (not the air temperature) is 60° F or above. Soil cannot be used to set plants or plant seeds until all odor of chloropicrin has disappeared, which may take 2 to 4 weeks depending on the soil temperature.

D-D MIXTURE: The killing agent here is a mixture of chemicals called dichloropropylenes and dichloropropanes. From the two prefixes "di" the trade name "D-D" was coined by its originator, the Shell Oil Corporation. D-D should not be confused with "DDT" or "DDD", which are insecticides. The trade name D-D is so well known that it will be used hereafter in this article when speaking of any dichloropropylene-dichloropropane mixture, regardless of the manufacturer. The cost of D-D mixture is much less than that of chloropicrin. Applied at rates of 20 to 25 gallons per acre, a price of from $40 to $60 per acre, including cost of application, can be expected. In Mississippi and other States custom operators inject the fumigant. The larger the area treated and the greater the ease of the application, the lower is the price. D-D mixture eradicates only nematodes though claims have been made that it will destroy some soil fungi. These are exceptional, specific cases, and only at extremely high dosages of the fumigant. In a few instances bacterial soil infestations have been eliminated by D-D mixture. This fumigant is relatively easy to handle. Its fumes are much less irritating and less volatile than those of chloropicrin; however, should it be spilled on the hands, they should be immediately washed with soap and water. If clothes or shoes become wet with D-D it is advisable to change them at once. Some people are more sensitive to D-D than are others.

No soil surface seal is necessary with D-D. The soil temperature can be relatively cool, 40° to 50° F and the fumigant will still be effective. Plants can be set out in treated soil in 2 to 2 1/2 weeks after treatment; the higher the subsequent soil temperature the shorter is this interval. Treatment of soil in fall rather than in spring eliminates the necessity for a waiting period before planting. Fumes of D-D do not cause injury to aerial parts of plants growing near treated soil but they will kill the roots of plants nearby (18 inches to 2 feet).

ETHYLENE DIBROMIDE: A more recent development than chloropicrin or D-D mixture, ethylene dibromide (abbreviated "EDB"), a third liquid fumigant, is relatively cheap. Prices for the fumigant, 6 to 12 gallons (85% active ingredient) per acre, vary from $36 to $50 per acre; to this should be added the cost of application, an additional $10 to $15 per acre depending on conditions. Ethylene dibromide is manufactured under several trade names, viz. Dowfume W-10, Dowfume W-15, Dowfume W-40, Dowfume W-85, Garden Dowfume, Iscobrome D, Soilfume 80-20, Soilfume Caps, etc. The percentage of active ingredient in an ethylene dibromide fumigant may vary from as low as 10 to as high as 85. The diluent is usually an inexpensive naphtha. Garden Dowfume has the lowest percent active ingredient. EDB is relatively easy to inject into soil and its fumes are of low toxicity to humans and animals. It is effective only against nematodes; it needs no surface seal of treated soil; soil temperatures should be 50° F or above; it disappears from soil rather slowly, in 2 to 3 weeks, depending on the soil temperature. Gelatin capsules, each containing the correct amount of ethylene dibromide (1/2 cubic centimeter) to fumigate one cubic foot of soil are available (Soilfume Caps). The capsules are placed in holes punched 6 inches deep in the soil, one hole per square foot, one capsule per hole. They work best in moderately moist soil which facilitates dissolving the gelatin capsule. Being relatively costly (1.5 to 2.0 cents each capsule), the use of capsulated ethylene dibromide is limited to small plots; however, the convenience of the method of apply-

ing the fumigant is great.

METHYL BROMIDE: Until recently, this odorless but very poisonous and highly volatile chemical (it boils at 40° F) was limited to the fumigation of granaries, packaged foodstuffs, nursery stock, and fresh fruits, for insects known to be or suspected of being present. It also is effective beneath a gasproof cover against nematodes and many weed seeds (1 pound methyl bromide per 100 square feet), but poorly so against fungi except in higher concentration (2 pounds per 100 square feet). Methyl bromide is hard to handle because of its low boiling point, but being odorless is not unpleasant to work with; a useful innovation has been the addition of 2% chloropicrin which acts as a warning of the presence of the very poisonous methyl bromide. It is expensive but not so costly as chloropicrin. Cost ($0.90 to $1.00 per pound in small quantities; $0.50 to $0.60 per pound in larger amounts) limits its use to land areas of high productivity value such as greenhouses or seedbeds. The prompt application of a surface seal is even more essential with methyl bromide than with chloropicrin. The seal may be gasproof paper, cloth, plastic, or even water (1 to 2 quarts per square foot of treated area). A plastic cover, 15 feet square, costs about $6, but it will last for 3 to 4 years if cared for properly. This fumigant is marketed under the trade names Dowfume G and Dowfume MC-2 (Dow Chemical Company), or Iscobrome I (Innis Speiden Co.). The Dow Chemical Company sells methyl bromide (MC-2) in sealed containers, with so-called "Jiffy" applicator equipment which includes a release mechanism and application tubing. It should be noted that Dowfume MC-2 is toxic to aerial parts of growing plants near to the area where it is used; this toxicity is due to the low percentage of chloropicrin present (see above). Methyl bromide is most effective at temperatures above 50° F. Soil may be used for seeding or for transplants within 4 to 5 days after treatment.

NEMAGON: This fumigant, 97% by weight 1,2-dibromo-3-chloropropane (DBCP) and 3% other halogenated C_3 compounds, made commercially available (as a liquid or absorbed on an inert powder) by Shell Chemical Co., about 3 years ago is effective only against nematodes. In its liquid form Nemagon is injected into the soil with the equipment used to apply the other fumigants previously described. Dosages as low as 1/2 gallon (17.3 pounds) per acre of the technical material, applied in rows 42 inches apart, have given excellent control of root-knot nematodes. Broadcast treatments, with the liquid injected in rows 12 inches apart, 1.0 to 1.5 gallons per acre, in some areas have been even more promising. For application the technical (97%) Nemagon is diluted with an aromatic hydrocarbon solvent, to give a 25%, 50%, or 75% Nemagon by volume, and may be used "as is", or be further diluted with mineral spirits or with kerosene. Nemagon Emulsifiable Concentrates also are available, in which the technical material is diluted with water before use to give desired concentrations. Nemagon in granular form, wherein the toxic material is absorbed on an inert carrier, contains as high as 50% by weight of Nemagon, but more commonly the formulations contain 10% or 25%. With 10% granular Nemagon, for a dosage of 1/2 gallon per acre use 86.5 pounds, for 1 gallon per acre twice as much, and so on. For a 25% granular Nemagon, the necessary quantities can be calculated. The granular Nemagon can be applied alone or it can be added to fertilizer and the combination applied in one operation. If broadcast, the granules must be plowed under immediately. Recent research, by experiment station workers and private industry, has shown that we probably can obtain adequate soil disinfestation (disinfection) with Nemagon applied only 4 inches deep instead of the customary 6-inch depth. Nemagon in its granular form must be stored in tight-fitting metal containers or it quickly loses its potency. It should be applied when soil temperatures, at a depth of 4 to 6 inches, are 50° to 80° F. As is true of other soil fumigants, the soil to be treated should be in good tilth and contain adequate moisture for good seed germination. Application should be made to soil 2 to 3 weeks before planting. However, Nemagon can also be applied as a drench to established lawns and turf or to perennial plantings of citrus, grapes, and a number of tree and small fruit crops as it has relatively low toxicity to the established roots of these plants. On most ornamental plants and turf 1 pint of Nemagon (75% concentration) is diluted with 100 gallons of water and applied to each 1000 square feet; if the 25% concentration of Nemagon is used, then 3 pints are diluted with 100 gallons of water. This is equal to 4 gallons of Nemagon (97%) per acre.

Nemagon is quite stable in the soil and is not easily lost following injection. Therefore, no cover is needed. It disperses slowly because of its high density and stability, and for this reason it takes 2 to 3 months to penetrate 3 to 4 feet. This residual action of Nemagon possibly could be disadvantageous in certain rotations.

Nemagon is relatively non-toxic to humans, but the liquid and its vapors can be harmful

if proper precautions are not observed. It seems to be in the same range of toxicity as D-D mixture, or slightly less. If Nemagon gets on the skin it should be immediately washed off with soap and water. Contaminated clothing and shoes should be immediately removed.

Nemagon costs about $20 to $25 per gallon (17.3 pounds), or 11 to 14 cents per pound, for the 97% concentration. The 10% concentration costs about 26 to 30 cents per pound in large quantities, considerably more in pound lots. To treat a lawn by drench costs about 1 cent per square foot. On a large scale, the cost of Nemagon is about $80 to $100 per acre, exclusive of cost of application.

VAPAM: This water-soluble soil fumigant, sodium N-methyl dithiocarbamate dihydrate, is a new (1955), temporary, soil sterilant effective without soil covering. It is said to be highly effective in control of weed seeds, nematodes, and fungi and also shows promise for control of several species of soil-infesting arthropods such as wire worms, the grape phylloxera, the garden centipede, and the bulb mite. Vapam has low toxicity to man and animals but is highly toxic to plants in the soil. It is available as a 4 pounds per gallon concentrate (Vapam 4-S), which decomposes rapidly in moist soil to release a penetrating gas which fumigates and then dissipates within a relatively short period of time. Vapam can be introduced into irrigation systems, sprayed as a concentrate in the plow-sole, sprinkled on the surface of the soil in dilute solution (1 pound diluted with 5 to 10 gallons of water per 100 square feet) followed by rotatilling, or by deep or shallow injection. Penetration and effectiveness are improved by a water seal (15 to 50 gallons of water per 100 square feet), but good results have been obtained without any seal. Vapam has been used in seedbeds, on turf and in home gardens. A dosage of 3/4 to 1 pound of Vapam per 100 square feet is required for a seedbed treatment. For preplant field fumigation for control of nematodes and fungi, a dosage of 25 gallons (100 pounds) per acre is suggested where the entire area is treated, and when application is made only to the rows, a dosage of from 10 to 12 gallons is indicated. The cost of Vapam is between 75 cents and $1.00 per pound.

Like other soil fumigants, Vapam is most effective in light-textured soils, at soil temperatures, 6 inches deep, from 40° to 80° F. Similarly, the soil to be treated should be in good tilth and of sufficient moisture content to insure good seed germination. At least 7 days, better 14 days, should elapse between application and planting.

Vapam can be used to sterilize potting soil by adding 1 ounce of Vapam 4-S to 3 1/2 cubic feet of soil in a cement mixer and mixing thoroughly. Or, the soil may be spread out in a 2-foot layer and sprinkled with 1 quart of Vapam 4-S, diluted to 1 to 2 gallons volume. As soon as the chemical has soaked in, the treated soil is sprinkled with a similar volume of water to act as a seal. On turf, use 1 quart of Vapam 4-S, diluted to 1 to 2 gallons with water which is applied with a sprinkling can to each 100 square feet. Vapam can find a use on soil in which noxious weeds and other pests are present and where it is desired to start a new lawn, new vegetable garden, new golf green, etc. Used at a 1:20 concentration diluted with water, the Vapam kills all vegetation in its path. It will move 3 to 4 inches laterally beyond the limit of its application and care should be taken not to injure valuable bordering vegetation.

Vapam and its decomposition products may be irritating to the eyes or skin. Avoid inhaling its mist or fumes. As with D-D mixture and with Nemagon, if Vapam comes in contact with the body wash it off immediately with soap and water, and contaminated clothing should be removed at once. Vapam should not be used in the presence of living plants unless adequate ventilation in maintained. Vapam is sold by the Stauffer Chemical Co., of New York City.

V-C 13 NEMACIDE: Another relatively new material is V-C 13 Nemacide whose active ingredient is O-2,4-dichlorophenyl O, O-diethyl phosphorothiate. It is comparatively nontoxic to plants, acts only as a nematocide and is usually applied as a water drench to turf, to the soil surrounding the roots of living plants or to soil as a preplant treatment. To aid in the dispersion of the nematocide when used as a preplant treatment the soil is either disked or roto-tilled after treatment. V-C 13 Nemacide can be mixed with a solid carrier, placed on the surface of soil in rows, or broadcast, and then roto-tilled into the top 6 inches. Application to nematode-infected (infested) plants also has been carried-out by introduction of V-C 13 Nemacide into irrigation water or overhead sprinkling systems.

A non-volatile liquid, V-C 13 Nemacide is offered for sale as a 75% emulsifible concentrate for application in a water emulsion. One gallon of the concentrate diluted with 25 to 50 gallons of water, is applied to 1600 square feet. The treated area is then well watered to wet the top 4 to 6 inches of soil thoroughly. No cover is necessary. On newly established turf, only 1/2 gallon of V-C 13 Nemacide per 1600 square feet should be used; on heavy soil, or

where large established root systems are being treated, 2 gallons of V-C 13 Nemacide per 1600 square feet are recommended. V-C Nemacide is residual in the soil and persists for several months. Apparently, the chemical acts by destroying the free nematodes and also the nematodes that subsequently emerge from infected roots. It may or may not destroy nematodes within roots.

V-C 13 Nemacide is hazardous if swallowed, inhaled, or exposed to the skin. It should be washed off immediately with soap and water. It costs between $25 and $30 per gallon (of the 75% emulsifible concentrate), that is, to treat soil with this fumigant costs between 1 cent and 1 3/4 cent per square foot. It is a product of the Virginia-Carolina Chemical Corporation of Richmond, Virginia. At present, V-C 13 Nemacide is not available through regular channels in Mississippi.

TERRACLOR: This fungicide, whose active ingredient is pentachloronitrobenzene (PCNB), is available from Olin Mathieson Chemical Corporation, Baltimore, Maryland, as a light buff to white, 75% wettable powder, or as a 10%, 20%, or 40% dust, or as a 2 pound per gallon emulsifiable concentrate. Relatively insoluble in water it is described as "the first powdered, non-volatile fungicide to be introduced." Because of its low water solubility and low volatility, Terraclor is safe for use in treating soil and plants at recommended rates, just prior to planting (or seeding), at time of planting, or as a foliage application.

One application, of as low as 30 to as high as 1950 pounds of 10% dust per acre, or at comparable rates for the 20% and the 40% dust, depending on cultural conditions and the type of pathogen to be controlled, often gives effective plant protection against soil-borne fungus pathogens from planting time to crop harvest. For the wettable powder, amounts of Terraclor that have been recommended range from as low as 2 to as high as 260 pounds per acre, and for the emulsifiable concentrate the quantities of Terraclor range from 1 1/2 gallons to 10 gallons per acre. Terraclor is said to be ineffective against the genera Fusarium, Pythium, Phytophthora, and Thielaviopsis, but is very effective against Rhizoctonia, Sclerotinia, Sclerotium, Plasmodiophora, Streptomyces, Tilletia, and Botrytis.

Terraclor can be applied as a dust or as a spray to soil, prior to planting or at time of planting seed or setting-out of transplants. Terraclor also can be used as an eradicant against fungus soil pathogens following crop harvesting. It is important that the Terraclor come into intimate contact with the pathogen in the soil; therefore it is recommended that the fungicide, if placed on the surface of the soil, be plowed under (depth of plowing ranges from 2 to 6 inches depending on normal cultural practices and habits of the pathogen), or applied in the furrow. It can be mixed with fertilizers and simultaneous application made. Under normal working conditions Terraclor does not cause irritation. However, the inhalation of dusts or spray mists should be avoided, for Terraclor may cause skin irritation to sensitive individuals. Hands and face should be washed well with soap and water following exposure.

MYLONE: This chemical, whose active ingredient is 3,5-dimethyltetrahydro-1, 3-5, 2H-thiadiazine-2-thione, newer to the soil fumigant list than either Vapam or V-C 13 Nemacide, is recommended for control of soil-borne fungi, nematodes, and weeds, particularly in tobacco and vegetable seed beds, ornamental propagating beds and in gladiolus culture. Mylone is available either as an 85% wettable powder that can be applied with a sprayer, or as a dry powder which is applied with a fertilizer spreader, and needs no cover or seal. It is used at the rate of 300 pounds (of the 85W) per acre (3/4 pound per 100 square feet). It costs about $8 to treat a 100-square yard bed. After application Mylone is mixed with the soil to a depth of about 6 inches, using a rototiller or disk. Best results are obtained if the treated area is moist. If possible, wet the treated-cultivated soil with 1 to 2 inches of water to retain the fumigant. No plastic cover is needed. Fourteen to 21 days later the soil can safely be used for crop production. Mylone is not a poison and it is not readily absorbed through the skin, nor is it a skin irritant. It is a product of Carbide and Carbon Chemicals Company, 30 East Forty-Second Street, New York City.

TELONE: This is the newest of the soil fumigants, exclusively a nematocide, and is a product of Dow Chemical Company, Midland, Michigan. It is available at present only for experimentation. Telone has been called "the first commercially available soil fumigant composed of undiluted technical dichloropropenes." (It will be remembered that D-D mixture and Dowfume N consist of dichloropropanes and dichloropropylenes, and that Nemagon contains dibromochloropropane). Telone is said to be effective against many kinds of nematodes, including cyst formers. It has little insecticidal value. This liquid nematocide is used as a

preplanting soil treatment. It is injected into soil in the usual manner, at the rate of 20 gallons (200 pounds) per acre in sandy and loam types of soil and double this amount when used in muck soils. Soil temperature can be between 40° and 80° F, with soil moisture about medium.

Telone is very irritating to the skin and eyes and great care should be taken in its use, for it can cause loss of vision.

DISCUSSION

It is five years since this article was first written, and changes in concepts of soil fumigation have occurred. At that time the fumigants in use were chloropicrin, D-D mixture, ethylene dibromide and methyl bromide. There was no fumigant available which could be applied to the roots of living plants to reduce or remove nematode infection. Today, such remedial treatment is possible with Nemagon and with V-C 13 Nemacide.

Soil sterilization, formerly possible only with the high-priced chloropicrin and methyl bromide, can now be done at materially reduced costs with Vapam and with Mylone.

Attention has been focused on controlling cyst-forming nematodes, such as the golden nematode (Heterodera rostochiensis) and the more recently discovered and appreciated soybean cyst nematode (Heterodera glycines). It has been pointed out that spot eradication treatments can be practical in the control of important pests such as these, even with the relatively high priced methyl bromide. Telone is mentioned as a potent eradicant of cyst-forming nematodes.

Ethylene dibromide, D-D mixture, Nemagon and V-C 13 seem to be running neck and neck in the choice of a relatively cheap nematocide that can be used without the necessity of a soil cover. Perhaps D-D mixture is being replaced to some extent by the same company's Nemagon. Methyl bromide still finds widespread use for treating valuable soil areas, such as seedbeds, to control all pests present, but Vapam and Mylone definitely should now be considered, since their performance is essentially similar to that of methyl bromide and they are appreciably cheaper.

Terraclor shows great promise of controlling those vigorous soil-borne fungus pathogens that attack growing plants, such as Rhizoctonia, Sclerotinia, and Sclerotium. Its low solubility in water makes it relatively long-lasting in its plant protectant and pathogen-eradicant effects.

The development of a fumigant that can be applied to the roots of living plants, particularly valuable perennials or established turf, to rid the plants of nematodes with little or no injury to the host plant, is a tremendous advance in nematode control. Fumigants Nemagon and V-C 13 Nemacide are receiving much attention in this respect.

A further significant advance in the application of soil fumigants has been the absorption of the liquid on powders, to produce an easy-handling granular form, which can be applied by a fertilizer spreader, separately or combined with the fertilizer. Broadcast surface application of the fumigant followed by disking or harrowing the soil is now possible and reduces costs. The ease of using such materials as Nemagon, V-C 13, Vapam and Mylone definitely is increasing the appeal of modern soil fumigants to the grower or home gardener. Instead of worrying about the application of a cover to treated soil to retain the fumes of the chemical, the home gardener or big farmer can sprinkle the soil with water, which acts as a pretty good seal.

While it still is recommended that a fumigant be introduced 6 inches below the surface of the soil, recent findings indicate that we may be able to get by with only a 4-inch depth of application. This would mean a saving in energy and therefore in money.

There has developed an increasing interest in the grower, the potential buyer, among the manufacturers of soil fumigants. Mississippi has lagged behind other States in this respect, probably owing to the fact that the values of soil fumigation have been difficult to demonstrate to the cotton grower. There are many other agricultural activities, however, such as turf and lawn grass, nursery stock, ornamental and possibly tree fruit production which, without the slightest doubt by this writer, are suffering considerable losses from attack by soil pests. Another fertile field for the soil fumigant salesman is the home garden. Most gardens are full of soil pests, particularly nematodes, but gardeners do little about them because of the trouble involved in treatment procedures and the relative unavailability of the fumigants.

MISSISSIPPI STATE COLLEGE

BRIEF NOTES ON PLANT DISEASE OCCURRENCE

WHEAT SNOW MOLD IN EASTERN WASHINGTON IN 1956-57

By Roderick Sprague[1]

In contrast with the devastation caused in 1955-56, there was no snow mold (Typhula spp. plus Fusarium nivale) in our plots in Douglas County, Washington in the spring of 1957. Growers who applied Ceresan M2X at 2 1/2 pounds per acre, either in heavy talc or in liquid, reported increased yields even though snow mold was absent. In Okanogan County, where some late mold developed, growers were satisfied with a dust of the Ceresan which one plane operator developed, using a fast-falling talc carrier. Ceresan 200 at 2 1/2 to 3 pints per acre was also used in Okanogan County with good results. Costs are still too high for universal use except in certain high altitude areas in Okanogan County where snow mold must be controlled every year.

At this date, in December 1957, prospects for snow mold in our present plots are uncertain. Some heavy early snows in October vanished soon. The ground was virtually bare on December 5, 1957.

WASHINGTON STATE COLLEGE, TREE FRUIT EXPERIMENT STATION, WENATCHEE, WASHINGTON

[1]Plant Pathologist, Tree Fruit Experiment Station.

THE "YELLOW CHLOROSIS" OF OATS RE-APPEARS IN THE PIEDMONT OF SOUTH CAROLINA

By R. W. Earhart[1]

Based on the reports obtained from our December small grain disease survey, and those from County Agents, the "yellow chlorotic condition" of oats, reported earlier[2], is present at this time in at least seven of the Piedmont Counties of South Carolina. These counties are: York, Chester, Newberry, Saluda, Laurens, Anderson, and Oconee. This chlorosis first appeared over a widespread area of the Piedmont in March and April of 1957. As was the case in the observations made last spring, it is manifested by a chlorosis of the leaf blades which ranges from a faint interveinal striping to a "golden" yellowing of the entire blade. The leading variety of oats currently being grown in South Carolina appears to be peculiarly susceptible to this particular chlorosis.

SOUTH CAROLINA AGRICULTURAL EXPERIMENT STATION

[1]Plant Pathologist, South Carolina Agricultural Experiment Station.

[2]Robert W. Earhart. Small grain diseases of South Carolina, 1956-57. Plant Dis. Reptr. 41: 863-870.

BLACK STEM AND LEAF SPOT DISEASE OF ALFALFA AND CRIMSON CLOVER

By Julius M. Elrod[1]

Alfalfa, seeded on land which has been in reseeding crimson clover for several years, may be severely damaged by summer black stem and leaf spot disease caused by Cercospora medicaginis Ell. & Ev. The disease attacks the alfalfa plants during the late summer and early autumn, often killing a large number of plants and reducing the stand.

This condition has been observed in a number of alfalfa fields seeded by farmers and in an alfalfa variety test seeded at the Georgia Experiment Station during the fall of 1956. Part of this test was on land which had been in crimson clover for several years and part on land where clover had not grown. On the first area, stands were reduced by the disease, whereas excellent stands developed on the latter area.

On the 18 varieties included in the test, Lahontan and Rhizoma were damaged most severely. Atlantic, Buffalo, Oklahoma Approved, and Williamsburg showed the least amount of damage. Narragansett, Ranger and Vernal had slightly more damage, and the disease was still more severe on the other varieties but not to the degree suffered by Lahontan and Rhizoma.

Dr. B. B. Higgins, Botanist Emeritus, Georgia Experiment Station, identified the disease.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

[1]Associate Agronomist, Georgia Experiment Station.

TWO NEW GEOGRAPHIC LOCATIONS FOR THE SUGAR BEET NEMATODE, HETERODERA SCHACHTII

By Fields E. Caveness[1]

Examination of soil and sugar beet samples from Imperial County, California, and Butte County, South Dakota, have revealed infestations of Heterodera schachtii Schmidt, 1871.

Two fields were found to be infested in Imperial County by the Beet Sugar Development Foundation survey, with more infestations located later by the California State Department of Agriculture. The H. schachtii population densities ranged from light to moderate.

Sugar beets and soil from a field in Butte County, South Dakota, were submitted to the Foundation by the Utah-Idaho Sugar Company. The sugar beet roots contained numerous cysts and a high larval population density was found in the soil.

These are the first known reports of H. schachtii from the geographically isolated Imperial Valley of California and the State of South Dakota.

BEET SUGAR DEVELOPMENT FOUNDATION, FORT COLLINS, COLORADO

[1]Nematologist, Beet Sugar Development Foundation.

ANNOUNCEMENT
TRAINING COURSE IN "AIR POLLUTION EFFECT ON VEGETATION"

Paul F. Woolrich, Chief, Air Pollution Training, sends the following announcement of a training course:

A three day training course, "Air Pollution Effects on Vegetation", will be presented at the Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio, during the period March 10-12, 1958. This program is intended for scientists, engineers, agricultural specialists and such others involved in assessment of air pollution effects on vegetation.

Included on the agenda are the following subjects:

Air Pollution and Agricultural Economics
Sampling for Foliar Analysis and Treatment of Data
Similarities Between Disease Symptoms, Insect Damage and Chemically Induced Injury to Plants
The Effects on Plants of: Sulfur compounds; halogens and halogen compounds; ozone, oxidants and smog; other gases and vapors; hydrocarbons; particulates; mixtures of gases; acids and acid gases.

Enrollment in this course is by application, and no tuition fees are required.

Further information and a detailed agenda will be sent on request. Such requests should be addressed to: Chief, Air Pollution Training, Robert A. Taft Sanitary Engineering Center, 4676 Columbia Parkway, Cincinnati 26, Ohio.